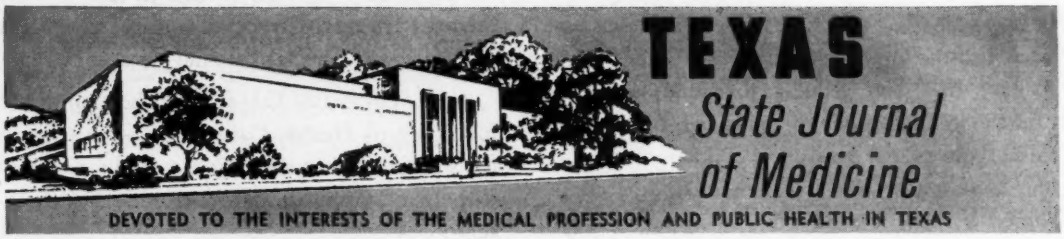

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House of Delegates Has Important Business

All members of the House of Delegates have received the Handbook for Delegates, which includes the annual reports of officers, boards, councils, and committees to be considered by the House. Action on these reports comprises the major portion of the business of the House, and the decisions reached on many matters may affect every member of the Texas Medical Association. For this reason sessions of the House are open to any member of the Association. Although such visitors may not participate in discussion or voting, they are urged to express to their county society delegates their views on any business coming before the House and to suggest other matters which should come to the attention of this policy-making assembly. Furthermore, any member may speak at hearings of reference committees on business to be voted on by the House.

Among the more important items to be presented to the House of Delegates at the 1957 annual session in Dallas later this month are the following:

Branch Societies.—The Board of Councilors is recommending that in some carefully selected instances, branch societies of component medical societies be allowed with the incorporation of several basic requirements. These are that a request come from the parent society by two-thirds majority vote, that a request come from 80 per cent of the members in the semi-isolated area desiring to become a branch society, that 25 or more members be required as the minimum to establish and maintain a branch society,

that only a member of the parent society may be a member of a branch society and disciplinary jurisdiction must remain in the parent society, that the request for establishing a branch society must be approved by a two-thirds vote of the Board of Councilors in regular session and such a request must be accompanied by the detailed plan of operation and necessary changes in the constitution and by-laws of the parent society, and that by two-thirds majority vote the parent society may abolish its branch society.

Abolishment of the Office of Vice-President.—The amendments proposed by the Council on Constitution and By-Laws to abolish the office of Vice-President are the result of careful consideration of the proposal made by Dr. F. J. L. Blasingame at the 1955 annual session that the Vice-President succeed to President-Elect, partly an effort to enhance the vice-presidency. Succession of Vice-President to President-Elect and ultimately President would mean three years of work requiring considerable time, money, and enthusiasm, which does not appear justifiable, the Council on Constitution and By-Laws believes. Various plans were considered at length by the Council before suggesting that if there is to be a change, the best solution would be to abolish the office entirely. Provision is made for a President pro tempore and the election of an interim President should the office become vacant. Constitutional changes are involved; therefore, final action cannot be taken until the 1958 session.

Restrictions on Intern, Resident, and Inactive Members.—Two Constitutional amendments introduced by the Harris County Medical Society at the 1956 annual session will be up for a vote at the coming meeting. They stipulate that intern and resident members and inactive members shall not have the right to vote, hold office, endorse applications for membership, or serve as a delegate or alternate delegate to the Texas Medical Association. A Constitutional amendment introduced by the Board of Councilors to restrict intern and resident members from the right to vote or hold office was passed at the last annual session.

Unit System Fee Schedule.—The Committee for Study of Health Costs is proposing that a fee schedule using the unit system be prepared to help major medical catastrophic health insurance work. Catastrophic health insurance was originated by Blue Shield in 1951, and in 1954 was extended to include cancer. The cancer coverage has been and is an experiment; it is being watched by other insurance companies all over the country. There is no question as to the need for this type coverage, but the payments of benefits are skyrocketing. The medical profession must make the coverage work, and to this end the Committee suggests that a unit fee schedule be prepared by the appropriate committee and distributed to each member of the Association as a possible guide in determining fees for service.

Code for Interprofessional Conduct.—The establishment of a code setting forth principles applying to interprofessional conduct and practices to serve as a guide to doctors and lawyers is being proposed by the Committee to Study Liaison with the State Bar of Texas. A copy of the Wisconsin code has been mailed to each member of the House of Delegates for perusal, it being implied that any code adopted in Texas would be basically similar to that.

Medicare.—The House of Delegates in special session on September 9 decided that the Texas Medical Association would participate in the military dependent medical care program. A progress report will be given at the annual session in Dallas, with further review then as indicated. Texas, like other states participating in the act, will be asked by the Office of Dependent Medical Care to extend its contract from July 1, 1957, until it has been renegotiated next year. (Five state contracts will be negotiated per month with Texas negotiations set for January.) All state medical societies are participating in the program, with two

exceptions, Rhode Island, where the Army is paying doctors directly, and Ohio, where the Army has contracted with an insurance company to handle the physicians' Medicare.

Numerous other matters contained in the many excellent reports printed in the Handbook will come to the attention of the delegates. For example, the Committee on Mental Health will report on Dr. Francis J. Gerty's review of the mental health picture in Texas; the Committee on Patient Care will give a progress report on the work of the Texas Commission on Patient Care; the Committee on National Emergency Medical Service will evaluate Operation Rebound; and the Committee on Maternal Mortality will give statistics on 1955 maternal deaths and the trend evident. These developments and many, many others reported in the Handbook and to be presented in supplementary reports will require attention.

Delegates are urged to review carefully the information contained in the Handbook and to discuss the various reports with their colleagues. All doctors should have an interest in the affairs of the Association; they are welcome to take advantage of this once-a-year opportunity to be present when policies are being made.

—HOBART O. DEATON, M. D., Fort Worth.

Top Scientific Program Available in Dallas

The most outstanding scientific program of the year in Texas is being offered this month, when the Texas Medical Association and 17 related specialty societies meet jointly in Dallas. The efforts of many physicians in arranging a strong educational meeting—including presentations by 24 noted out-of-state guest speakers acquired cooperatively, other renowned special speakers from in and out of Texas, and Texas physicians—are expected to prove well worth while as the annual session begins.

All scientific meetings in Dallas are open to members of the Texas Medical Association. Not only will the nine scientific section meetings, general meetings, refresher courses, motion pictures, and exhibits be of interest to the doctors in attendance, but equally important are the sessions of the related organizations. Beginning on Saturday and continuing through Tuesday, with the majority being scheduled for Sunday, related specialty society programs deserve attention.

Specialty societies, in addition to utilizing the majority of the guest speakers, include on their programs presentations by such medical experts from out of Texas as Dr. Buford H. Burch, chief of thoracic surgery, Patton State Hospital, Patton, Calif. (Texas Chapter, American College of Chest Physicians); Dr. Harris Isbell, director, Addiction Research Center, U. S. Public Health Service Hospital, Lexington, Ky. (Texas Neuropsychiatric Association); Dr. J. Roderick Kitchell, associate professor of cardiology, Jefferson Medical College of Philadelphia, Philadelphia, Pa. (Texas Heart Association); Dr. Philip M. Markle of the American Board of Aviation Medicine, Memphis, Tenn. (Texas Air-Medics Association); Dr. Herman J. Moersch, president, American College of Chest Physicians, Rochester, Minn. (Texas Chapter, American College of Chest Physicians); and Dr. Jackson A. Smith of the University of Nebraska, Omaha, Neb. (Texas Geriatrics Society).

Other groups than those 17 actually designated by the Council on Scientific Work as related organizations also are doing their share to make the annual session a success. The Texas Division of the American Cancer Society and the Texas Pediatrics Society are helping to bring several guest speakers to the convention; the Texas Chapter of the American Academy of Pediatrics is cooperating with the Section on Pediatrics in the presentation of a panel discussion on accidents and poisonings in childhood; the Texas Physical Medicine and Rehabilitation Society is holding an organizational meeting; and the Fourth District of Texas

Chapter of the American College of Surgeons is having a session.

The American Medical Association's Committee on Aging will have a regional conference immediately preceding the annual session, under the chairmanship of Dr. Henry B. Mulholland, Charlottesville, Va., who also is a guest speaker. His schedule, typical of other guests' assignments, is an example of the cooperative spirit of the program planners; and his assignments include a talk before the Texas Diabetes Association, a refresher course, an address at a joint luncheon of the Texas Geriatrics Society and the Section on Public Health, and participation in a panel discussion for the Section on Internal Medicine. Dr. Wingate M. Johnson, professor of internal medicine, Bowman Gray School of Medicine, Winston-Salem, N. C., and a member of the AMA's Committee on Aging, also is participating in the scientific program.

Every effort has been made by the Council on Scientific Work, section officers, officers of related organizations, and others to assure the highest quality scientific program possible for the doctors of Texas at their 1957 annual session; and plans will begin in Dallas for the 1958 program. It is hoped more and more Texas physicians will follow in the footsteps of those who make it a practice to attend their convention yearly.

Texas Insurance Ranks High

The disability insurance plan sponsored by the Texas Medical Association compares favorably with other such plans offered through state and large county medical societies throughout the country. A review of 35 group disability insurance programs has been completed by the Insurance Committee of the California Medical Association. (See page 294.)

It is evident at a glance that Texas doctors have as flexible and as comprehensive a program available to them as is offered by any other medical group studied. The Council on Medical Economics and the Board of Trustees are to be congratulated for their part in providing such insurance.

★ Current Editorial Comment

Medicine and the Ministry

There is increasing recognition these days that the properly trained minister belongs on the health team—an insight which primitive culture sensed in making the "Medicine Man" and the "Holy Man" one person. An authentic insight is involved in the fact that the words *health, hale, whole,* and *holy* all come from the same Anglo-Saxon root. In the long run, the minister and the doctor are talking about the same thing.

The modern discovery, now that the interim conflict between science and religion is over, is that, as one famous surgeon once put it, "the body and soul live so closely together they catch each other's diseases."

Health, then, is not only a matter of the body, it is also a matter of the spirit.

Any doctor knows that the medical skill which can restore a man to physical fitness is sometimes relatively futile and that the man's physical recovery may be short-lived, if the man himself continues to suffer from a sense of emptiness, from a lack of anything to live for, or from a terrible sense of guilt. When a man's reason for living is gone, when death seems to him more desirable than life, when he cannot any longer live in peace with his betrayals, his needs from a health standpoint cross over the line from what is purely medical to what may, in essence, be primarily religious.

Excerpts from an address presented at the inaugural banquet of Tarrant County Medical Society, Fort Worth, Texas, January 8, 1957, by Dr. Walker, for 14 years minister of the University Christian Church in Fort Worth and current president of the Seventieth Annual Texas Convention of Christian Churches.

This department of the Journal presents editorial comment on current items pertaining to the science, art, and practice of medicine, contributed by members of the Texas Medical Association and others closely associated with the medical profession of Texas. Contributions not to exceed 500 words are invited.

The whole man—body, mind, and spirit—is sick, and his illness should be dealt with in terms of his wholeness and not merely in terms of his physical symptoms. In such situations, the minister's presence on the health team may be decisive. Admittedly he is not the quarterback on that team, but now and again it is his responsibility to kick the extra point which may decide whether the game is won or lost.

What I mean to point out is this: Ministers trained in modern seminaries undergo specific preparation for their role in dealing with sick people, taking courses usually taught by medical and psychiatric personnel. Because a great deal of our time is taken up with counseling people in various stages of physical or mental health, we need to know enough to know when we should send these people to you physicians. Sometimes religious problems are accentuated by feelings of physical inadequacy, so that medical care may be helpful in straightening out a religious or emotional problem. Sometimes counseling reveals neurotic or even psychotic tendencies, which the minister must learn to suspect so that he will know when to turn to a psychiatrist for help. For all of this he is trained in the seminary by medical people.

But the minister's role is more affirmative than that. He often has a function to perform even in "routine" illness—which, however simple it may appear to you, does not always so appear to the patient. There are times when the patient will share his fears and anxieties with his minister when, for various reasons, he will not do so with his doctor. He may not want the doctor to know how frightened he is at the prospect of major surgery, because to admit his fear to the doctor might be interpreted as lack of confidence in him. And, indeed, his fears may root occasionally in that very lack of confidence. In such situations, the best friend the doctor has is the minister who enters the sick room and honestly reassures the patient that he is in the best possible hands, and then, in a moment of prayer, commits *his* illness and *your* skill and wisdom to the hands of God. You are a better doctor when your patient is not afraid.

There are other functions which the minister has to perform in relation to sick people. I am sure a busy doctor going from one heavy responsibility to another among his patients sometimes wishes he did not have the patient's family to deal with. For a family's apprehensions and impatience most certainly can affect the patient's progress. A minister's best contribution may be dealing with these folk.

Still another situation in which work of doctor and minister is related occurs when the doctor, having done all that he can, is obliged to report to the patient that he must live out the rest of his life with a certain degree of permanent incapacity. Here it falls largely to the minister to help the patient build a new and meaningful life under the conditions of his handicap. The doctor, to be sure, defines the limits within which the life is to be lived, but getting the patient to accept the situation with creative grace is something which falls largely within the realm of the patient's religion and philosophy of life—the very realm where the minister is most at home.

The one function of the minister with reference to the sick which has always been acknowledged is his role in dealing with a terminal illness. Then, not only the patient, if he is to know this verdict, but the patient's entire circle of loved ones become the special concern of the minister. If the patient is to die, it is the task of religion to aid him to die nobly and to aid the loved ones to accept his death without bitterness.

Unquestionably, the art of healing requires a broad spiritual approach, as well as a broad medical one. I am almost prepared to say that no one is properly equipped to deal with ill people whose reservoir of spiritual insight and whose capacities for human kindness and understanding are depleted.

—GRANVILLE T. WALKER, B.D., D.D., Ph.D.,
Fort Worth, Texas.

♦ Dr. Walker, University Christian Church, Fort Worth.

President's Page

IT DEPENDS ON YOU!

The public's opinion of the medical profession is largely a composite of the public's opinion of individual doctors. Therefore it is up to me—and to you—to serve faithfully, capably, sympathetically, and reasonably every day, as a physician and as a citizen.

The medical profession and the public look to the pronouncements of Hippocrates as the origin of the ethics and principles of service to humanity, which place doctors high in the esteem of their fellowmen, but Hippocrates just restated the essentials of the Golden Rule as enunciated by the first Great Physician, Jesus of Nazareth.

It is entirely within the province of the Golden Rule for physicians to be good citizens—and as such to participate actively in their communities' churches, chambers of commerce, service clubs, boards of directors of business enterprises, and politics, if by politics we mean the exercising of the privilege of voting for proper officials.

Doctors are truly partners in health with many: fellow physicians; health departments; dentists, pharmacists, nurses, and others working in paramedical professions; medical schools; health phases of public schools and colleges; the good ladies of the Woman's Auxiliary; newspaper men and women; health committees of chambers of commerce and service clubs; and lay groups interested in certain diseases as cancer and tuberculosis. It is only as we shoulder our full responsibilities and opportunities of all-'round community service that we will continue to merit the esteem and real affection of the public.

And it all starts in my everyday work in my own office and radiates out from there—as is also true of you. Let's be faithful in service not only to our patients but also to our communities.

JUSTICE FOR 10,000,000 SELF-EMPLOYED

Physicians have a definite stake in the Jenkins-Keough bills now pending before Congress, which would permit self-employed individuals to participate in restricted retirement or pension plans, with certain income tax benefits or savings. For years workers in industry

and even officers of corporations have been able to have part of their incomes put into retirement or pensions, with such deposits being deductible in computing income taxes, while 10,000,000 citizens—physicians, dentists, lawyers, architects, insurance and real estate agents, smaller merchants, and others—have been penalized by being self-employed and therefore denied certain tax privileges enjoyed by corporations.

The American Thrift Assembly is being launched to get these 10,000,000 self-employed individuals informed and activated to personal contacts with Congressmen and later to systematic thrift by annual investments in retirement programs.

Write your Congressman and Senators STAT and let them know of your interest. Then when you hear of an American Thrift Assembly in your community, attend and get still better informed.

APPRECIATION FROM THE HEART!

Your President has enjoyed thoroughly the privilege of chatting with you informally through the President's Page of the *Journal* this year, and he thanks you for being tolerant and patient.

He appreciates deeply the wonderful cooperation he has had from everybody: the central office staff; the Trustees, Councilors, and other state officers; the legal counsel; the 300 faithful members of councils and committees; the officers and members of the Woman's Auxiliary; paramedical groups; the press of the state; members of the Legislature; his own family; and countless others.

And particularly is he grateful for the fine spirit of the best partners a doctor ever had—Drs. Cecil O. Patterson, Herbert A. Bailey, F. Clark Douglas, and John W. Fisher. Frankly, without their wonderful understanding and cooperation your President would have had little time to accomplish anything—and he is humbly and deeply appreciative to everybody.

Gratefully,

Milford O. Rouse, MD

ORIGINAL ARTICLES

Prevention and Treatment of Hemolytic and Allergic Blood Transfusion Reactions

WAYNE M. MEYERS

Houston, Texas

IN ITS BROADEST SENSE, the term "transfusion reaction" refers to any untoward change in the patient following a blood transfusion. Thus such varied factors as the type of anticoagulant used, emboli production, introduction of an infectious agent, or rate of infusion may be considered as possible sources of transfusion reactions. However, in this discussion, only those responses of the patient referable to a post-transfusion hemolytic reaction or the presence of an allergic state are considered.

Wayne M. Meyers, a junior at Baylor University College of Medicine, last year won second prize in the Schering Award contest for medical students in the United States and Canada with an enlarged draft of this paper.

As recently as 1951, blood transfusions carried mortality rates higher than anesthesia or appendicitis. With elucidation of the causes of transfusion reactions and the use of exacting laboratory techniques, reactions from incompatible blood have decreased. Allergic reactions may be reduced by noting the past history of the recipient and the donor.

Starting in 1900, Landsteiner's studies on agglutinating and hemolytic reactions between samples of human blood gave some explanation for the grave consequences of blood transfusion which had plagued medical practice for several centuries.[37, 38, 39] Landsteiner's investigations culminated in an understanding of the "Individual Differences in Human Blood," for which he was awarded a Nobel Prize in 1930.[40] Thus the ABO blood grouping procedure was established, and some other individual variations in human bloods were noted. The solution of reactions in ABO compatible bloods, however, still remained. The most notable of the intragroup systems, the Rh system, was established by Landsteiner and Wiener.[41] Previously Levine and Stetson had detected the presence of this entity and indicated its possible clinical importance.[45] Elucidation of the causes of the small percentage of reactions which take place in supposedly compatible blood transfusions is still a matter of much concern. As recently as 1951, it was estimated that blood transfusions carried mortality rates higher than anesthesia or appendicitis.[1] Increased transfusion demands in disaster situations (for example, war) have stimulated interest in methods of reducing transfusion hazards.

HEMOLYTIC REACTIONS

Post-tranfusion hemolytic reactions arise as a result of the interaction of erythrocyte antigens with antibodies (hemolysins) in the presence of complement. Usually, antibodies from the patient's plasma combine with the erythrocyte antigen(s) of the donor; however, the reverse situation may obtain. The former is seen most often since the concentration of antibodies in the donor's plasma is usually too low to produce a noticeable reaction on dilution in the recipient; if the antibody concentration of the donor is sufficiently high after dilution, hemolysis of the recipient's cells occurs. Kabat[31] has indicated that as few as 400 antibody molecules may sensitize a single erythrocyte. Wiener and Wexler[66] have introduced the concept that an agglutinogen may be composed of various blood factors.

The occurrence of A, B, and O substances in the human red cell wall has been amply demonstrated; A and B substances have been characterized chemically as polysaccharides.[69] Group O red cells, once thought to be peculiar in that they contained neither A nor B substance, recently have been shown to con-

tain a definite O substance.[68] Additionally, anti-O isoagglutinins have been detected in rare instances in A_1 and A_1B blood.[52] Normally, the plasma of group A individuals contains isoantibodies to B substance and vice versa, while group AB blood contains isoantibodies to neither A nor B substance; group O blood contains isoantibodies to both A and B substance. Thus, requirements for intravascular hemolysis may be met when blood of the improper group is transfused.

The universal donor concept was deduced from the distribution of antigens A and B and their isoantibodies. A group O person is a universal donor since O cells contain neither antigen A nor B and hence are not hemolyzed when transfused into persons of any blood group. However, the use of group O blood universally entails hazards. Apart from the content of possible intragroup isohemagglutinins, the isoantibody titer of the donor's plasma is of importance; for example, if anti-A exists in the donor's plasma in high titer, the erythrocytes of a group A recipient may be lysed. By adding a few milligrams of purified blood group substances A and B to a pint of blood, Witebsky and others[68] were able to reduce isoantibody titers, a procedure which may be useful where only high isoantibody titer O blood is available. Witebsky's experiments revealed differences in the nature of normal anti-A isoantibody and the antibody produced by previous contact with group A substance (pregnancy or transfusion). This latter isoantibody has been called "immune type" and is reminiscent of the incomplete Rh antibody in that a serum milieu is essential for agglutination. Subtype A_1 cells seem to adsorb the "immune" antibody selectively. Immune isoantibodies are considered especially dangerous even in low titers.

Davidsohn[18] has suggested the following procedure when universal donors are used: reject donors having isoagglutinin titers higher than 1:100 or 1:200; determine the presence of immune isoagglutinins and reject blood containing them regardless of titer. Use of A and B substances to reduce isoantibody titers is supported by Davidsohn, but should be reserved only for emergencies, since immune isoagglutinins are not removed and immune anti-A or anti-B may be formed in recipients. Severe hemolytic reactions such as the one reported by Elliott[15] in which O blood with an anti-A titer of 1:32,768 was used could have been averted by applying Davidsohn's principles.

Studies by Crosby and Howard[10] on casualties in Korea requiring large transfusions showed that universal donor blood may not be exceedingly dangerous. No hemolytic reactions were observed in 37 cases transfused with large amounts of universal donor blood. Not only from a study of casualties cared for under their supervision but from a review of statistics from World War II, Crosby and Akeroyd[9] were unable to demonstrate that the use of universal donor blood of low isoantibody titer caused renal injury from hemolysis. Although some hemolysis was detected, attributable to transfused antibodies, "no reactions were encountered and none were heard of in Korea that might have been ascribed to a dangerous universal donor."[9] Since no blood with an isoantibody titer higher than 1:256 was used, the usefulness of selected group O blood in emergencies without serious consequences was demonstrated. In the transfusion of 10,000 pints of group O, low titer, banked blood in Korea, Howard[28] reported only 3 hemolytic reactions. This is interesting since no crossmatch was attempted because of the urgency of the situation and since often 15 to 20 pints were administered. In the future, reactions due to isosensitization may be expected rather frequently in these persons.

Normal isoantibodies to the Rh-Hr system are not known; however, immune isoantibody production in an Rh-negative recipient by transfusing Rh-positive blood occurs often. Such recipients upon subsequent transfusion with Rh-positive blood may suffer a hemolytic reaction. The Rh-Hr system factors vary in antigenicity: Rh_0 (British "D") is the most antigenic and hence responsible for most intragroup hemolytic reactions (approximately 97 per cent). Clift[7] reported a severe hemolytic reaction due to anti-c indicating that the indiscriminate use of Rh-negative blood may be exceedingly dangerous. A similar case was presented by Frick[22] in a woman who had borne 13 children (6 were carriers of "c" antigen). Routine tests did not detect incompatibility in either of these cases. The importance of using sensitive techniques is thus emphasized when recipients with histories of transfusion or pregnancy are being transfused. Isosensitization to the Rh factors other than Rh_0 may produce only mild reactions resembling pyrogenic reactions.[65] Wiener indicated that isosensitization of the Rh-positive individual occurs.[64]

Table 1 lists Rh-Hr and other factors in order of decreasing clinical importance. Rosenfield[53] has made the following statement concerning the various blood factors: "In all probability, only Rh-Hr, K, Fy^a, M, S, s, Jk^a and Fy^b antibodies are capable of causing severe reactions and of these only Rh-Hr, K, Fy^a, Jk^a, S and M have been proved to do so. The P, Le^a, Le^b, 'cold' M, A_1, B_1 and O antibodies are best considered as the cause of confusion in performing crossmatches, rather than as being capable of causing severe transfusion reactions." Prevention of hemolytic reactions depends upon the detection of incompatibilities between donor and recipient. In recent years the incompatibility tests have been considerably refined; however, unexplained hemolytic reactions still occur. Strict attention to the techniques employed and knowledge of their limitations would reduce the in-

cidence of hemolytic reactions markedly. Discombe[14] has estimated that there is one hemolytic reaction with no apparent cause in 2,300 transfusions. Most group incompatible transfusions are avoidable; however, false reactions do occur in the laboratory.[29]

TABLE 1.—*Human Blood Factors Demonstrable by Isoantibodies.*

Factors	Alternate Names	Per Cent of Positive Reactions in White Population*		Comments
D–d	Rh_o–Hr_o	85	63	Anti–d very rare.
C–c	rh′–hr′	73	80	Anti–C rare in Rh–.
C^w	rh^w	1		
E–e	rh″–hr″	30	97	Anti–E more frequent.
K–k	Kell-Cellano	8	99.8	Becoming increasingly important.
Fy^a–Fy^b	Duffy	66	82	One example of anti–Fy^b.
Jk^a–Jk^b	Kidd	77	73	One example of anti–Jk^b.
S–s		56	88	One example of anti–s; numerous examples of anti–S.
M–N		79	71	Anti–N very rare.
Tj^a	Jay	Slightly less than 100		8 examples of anti–Tj^a.
Le^a–Le^b	Lewis	18	72	Associated with ABO secretor status.
Lu^a	Lutheran	8		Of questionable clinical importance.
P		75		No clinical importance.

*In each case in which the factors are paired, for example, D–d, the two values give the incidence of reactions for D and d, respectively.

Modified from Levine, P.; Koch, E. A.; McGee, R. T.; and Hill, G. H.: Rare Human Isoagglutinins and Their Identification, Am. J. Clin. Path. 24: 293 (March) 1954.

Intragroup incompatibilities are not always easily detected, and hence considerable work has been done in this area. Table 1 lists the important antibodies to be expected. There are doubtlessly unknown blood factors and isoantibodies, but these should prove to be exceedingly rare and of less importance statistically. Attention must be focused in this area if the small percentage of unexplained reactions is to be reduced. With the present knowledge of blood factors, more than 50,000 combinations are possible; a single blood sample has been known to be incompatible with as many as 1,000 different samples.[44]

Standard laboratory methods are described in several manuals.[2,50] An important development for the detection of factors involved in hemolysis due to Rh-Hr was the Coombs antiglobulin test.[8] This is a distinct method to detect incomplete antibody (variously called blocking, univalent, or hyperimmune). Recently this test's limitations have been studied by Hill and Haberman.[27] The Coombs test may give false positive reactions due to insufficient adsorption, cold agglutinins, or bacterial contamination; false negative reactions result from insufficient washing of sensitized cells, presence of agglutinoids in the antiglobulin, excessive amounts of anticoagulant, soluble antigens such as Le^a, old blood, bacterial contamination, or extended incubations following addition of antiglobulin. These authors suggested methods for the detection of these false reactions. Morton and Pickles have introduced a highly sensitive trypsin-treated cell method for detecting complete and incomplete antibody.[51] Table 2 summarizes the reaction of both antibody types.

The following steps have been suggested by Unger[59] for routine Rh-Hr determinations: (1) Rh-testing, (2) Rh-Hr typing, (3) tests for sensitivity for Rh-Hr antibodies, and (4) identification of the specificity and titration of Rh-Hr antibodies. Rh typing is carried out on the blood of each Rh_o-negative donor by the rapid slide method. Donors positive for Rh_o variant, rh′ and rh″ factors, are classified as Rh-positive to avoid isosensitization. Rh-Hr typing is performed on all persons who have had transfusion reactions or when crossmatching is difficult.

Rare incompatibilities have been determined by Levine and others[43] using a panel of six selected test cells. Where transfusion difficulties occur, blood from several donors is tested with the appropriate serum. Table 3 shows a representative test panel for the identification of the rare antibodies.

TABLE 2.—*Varieties of Rh-Hr Antibodies.*

Method of Testing	Bivalent	Univalent
Saline agglutination	+	—
Albumin-plasma conglutination	+	+
Enzyme treated cell	+	+
Antiglobulin		+

TABLE 3.—*Antigens in Panel of Group O Test Cells for Identification of Antibodies.*

Rh Antigens Most Likely Genotype		Other Antigens							
		MN	Kk	Ss	Jk^a	Fy^a	Le^a	P	Lu^a
R^1R^1	DCe/DCe	MN	O+	++	O	O	O	O	O
$R^1{}^wr$	DC^we/dce	N	O+	O+	+	+	+	+	+
R^2R^2	DcE/DcE	N	O+	O+	O	+	O	O	O
r′r	dCe/dce	M	O+	+O	+	+	O	+	+
r″r	dcE/dce	MN	O+	++	O	+	O	+	O
rr	dce/dce	M	+-	O+	O	O	O	O	O

After Levine, P.; Koch, E. A.; McGee, R. T.; and Hill, G. H.: Rare Human Isoagglutinins and Their Identification, Am. J. Clin. Path. 24:297 (March) 1954.

Anti-c, anti-e, and anti-K are considered the most important rare antibodies and commercial panel test cells are available for their detection. Rosenfield[53] has described techniques for "detecting every human isoantibody known."

These tests are aimed at one immediate goal—to find a donor whose red cells lack a blood factor for which the recipient possesses an isoantibody. Additionally, it is important to prevent the injection of a blood factor which the recipient does not possess in order that he will not become isoimmunized. Many cases are on record in which a person has become sensitized to a multiplicity of blood factors. Thus the recipient's serum should be checked before each transfusion if some time elapses between transfusions, since there is always the possibility of the recipient becoming isoimmunized in the meantime.

The aforementioned procedures in principle constitute the only prevention for hemolytic transfusion reactions.

TREATMENT

Treatment of transfusion reactions varies according to severity and is prompted by the clinical symptoms. The effects seen along with variations in blood volume administered have been studied and tabulated.[14]

The symptoms of the patient transfused with incompatible blood have been variously described, Denis' description given in 1667 being a classic.[54] Stevens[56] has described the hemolytic reaction concisely:

> . . . Patients experience a full feeling in the head, headache, severe chill with fever, and often nausea and vomiting. Usually there is bilateral flank pain. Shock and orthostatic hypotension are not so outstanding. The patient may after a short time return to a feeling of well-being, but the danger is not over until it is demonstrated that the kidneys have escaped damage.

Free hemoglobin must be demonstrated in the patient's serum to substantiate a hemolytic reaction

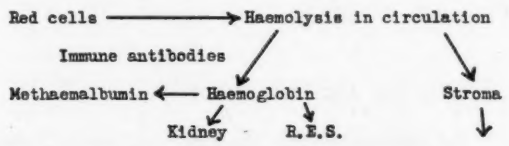

Fig. 1. Diagram showing the fate of intravascular hemoglobin. [Modified from Walsh, R. J.: Blood Transfusions and Acute Renal Failure, Med. J. Australia 1:219 (Feb. 12) 1955.]

diagnosis. The fate of intravascular hemoglobin has been diagrammed by Walsh[61] (fig. 1).

Hemoglobin release in acute renal failure following hemolytic transfusion reactions is of questionable importance.[14] Discombe's conclusions are that hemoglobin excretion is virtually innocuous; most patients subjected to hemolytic reactions are unlikely to develop anuria no matter how much incompatible blood is received; a few with some biochemical disorder causing renal function impairment (for example, dehydration or alkalosis) may develop anuria without shock or hypotension and may die. A few may show an allergic response, develop hypotension, anuria, and die within a few hours or several days. (Such allergic responses need not be concerned with blood factor systems, but may be related to some other component in blood.) The allergic hypothesis with the kidney as the shock organ is attractive since in paroxysmal hemoglobinuria the release of massive amounts of hemoglobin does not produce the symptoms sometimes seen in hemolytic reactions. Again this concept is tenable from studies conducted on the pathogenesis of black water fever. Quinine treatment is considered to be a precipitating factor in the ensuing anuria, its role being haptenic in nature. Fischel[18] has raised some provocative questions against the allergic basis of nephrosis, especially as regards the levels of serum complement as a criterion.

That severe renal damage may follow the administration of only small amounts of incompatible blood indicates the importance of the individual response; conversely, the transfusion of 1,500 ml. of incompatible blood may produce no renal damage, only jaundice.[16] The essential pathologic picture of the kidney in fatal post-transfusion hemolytic reactions is (1) stromal edema with cellular infiltration; (2) necrosis of epithelium of the ascending limb of Henle and of the distal convoluted tubules; (3) tubulovenous anastomoses; and (4) formation of heme-pigment casts.[14]

Upon diagnosis of a hemolytic reaction by detecting free hemoglobin in the patient's serum, the following additional tests have been suggested by Davenport:[12]

1. Serum bilirubin, urine urobilinogen, urobilin.
2. Azotemia, Cl^-, $Na+$, $K+$, in the event of urinary suppression.
3. Carbon dioxide combining power.
4. Urine acid-base reaction.
5. Examination of urine sediment.
6. Kidney function and, in some cases, liver function tests.
7. Hematologic studies.

Although sodium lactate or sodium sulfate is indicated by some[12] to relieve shock and hypotension, it is considered hazardous by others[14, 47] owing to ionic disturbances resulting in cellular dehydration. Fatal renal failure is too infrequent to warrant jeopardizing all anuric patients with such agents. Evans[16] has indicated that he does not agree that sodium sulfate is harmful, but that it may serve as a diuretic. The production of alkaline urine is considered by many to be

unimportant in preventing anuria. Administration of compatible blood, dextran, or plasma may relieve the shock and hypotension, and since these colloids are retained in the blood stream their action helps maintain blood volume. Vasopressors such as noradrenalin and methylamphetamine are considered useful in relieving peripheral dilatation if hypotension persists, the latter perhaps being more useful in maintaining better kidney circulation.[14]

The supportive treatment given anuric patients is the most important factor in recovery or death. Fluid and ionic balance are of utmost importance. These factors recently have been evaluated on both patients and dogs by Hamburger and Mathe,[25] and the following information is taken from their work. An accurate indication of water need is given by the symptoms and osmotic pressure of the plasma—for example, thirst and high osmotic pressure indicate a need of water. The diet should be salt free; however, care should be exercised since in untreated cases cellular overhydration and extracellular dehydration usually follow. In such cases besides fluid restriction, intravenous hypertonic saline solution may be given with caution to prevent pulmonary edema. In severe acidosis citrate, bicarbonate, or lactate solution may be used with the saline. Extrarenal dialysis is advised in extreme cases to remove fluid. The use of Bull-Borst regimen[4, 6] for anuric patients may be advisable.

Immediate exchange transfusions following hemolytic reactions are advocated by many, the rationale being that the incompatible red cells are removed.[32, 33, 36] Walsh[61] has expressed the belief that exchange transfusion to remove incompatible red cells may be both impractical and useless: impractical because of the large volume necessary (to be 90 per cent effective in exchange, 11 L. of blood are necessary for the average person) and useless because by the time exchange transfusion is initiated the hemoglobinemia has taken place and the unlysed cells destined to harmless destruction. Exchange transfusion may correct a hyperpotassemia following intravascular hemolysis, since sudden hemolysis may liberate sufficient potassium to cause cardiac arrest.[62] Renal impairment adds to the likelihood of potassium intoxication. Other methods are also recommended for the correction of hyperpotassemia: peritoneal dialysis, intestinal dialysis, use of the artificial kidney, or ion exchange resins.[61]

ALLERGIC REACTIONS

Allergic and pyrogenic reactions to transfusion are sometimes considered together; however, the allergic reaction will be emphasized here. Kilduffe and DeBakey[34] have divided this type of reaction as follows: (1) relatively mild, characterized by urticaria, slight angioneurotic edema and eosinophilia; (2) moderately severe, typified by difficulty in breathing, asthmatic rales, and involuntary defecation and micturation; and (3) severe, resembling anaphylactic shock which occurs during or soon after the transfusion. The incidence of allergic reactions is difficult to assess, primarily because of the variations in methods of recognition by different observers. It is roughly estimated as occurring in approximately 1 per cent of all transfusion reactions.[49] Even though these reactions are generally relatively mild, they should be considered seriously. Allergic reactions are often important because this insult on the patient, in addition to the condition for which transfusion was indicated, may produce grave results. Reactions of similar degree in healthy persons most often would be of little consequence.

The mechanism of allergic reactions is obscure, in contrast to the well delineated hemolytic reaction. Until this is solved, their effective prevention and treatment are unsure. It is generally assumed that the allergic reaction results from antigen-antibody combination.[5, 11] This combination may cause the formation of histamine or histamine-like substances which are physiologically active. Serum proteolytic enzymes have been implicated tentatively in the release of histamine by Gell[23] and others. The proenzyme plasminogen has been isolated from serum in a reasonably pure state.[35] This enzyme is activated by certain kinases,[58] and there are natural inhibitors present in human serum. The combination of antigen and antibody and the resulting complex may either reduce antiplasmin concentration or play some role in the actual activation of plasminogen. Such a theory is attractive, but there is evidence that plasmin can be injected into human subjects without producing allergic symptoms regularly.[19] The fact that known trypsin and plasmin inhibitors (for example, soybeans) may be allergenic does not obviate this theory since they would be acting as antigens to "trigger" the protease system while the concentration may be much too low to act as an effective antiplasmin. Other explanations of basic allergic mechanisms at the present are inadequate.

PREVENTION AND TREATMENT

The prevention and treatment of allergic transfusion reactions are on a somewhat empirical basis. Measures should be taken to prevent both the sensitization of recipients to foreign materials and the passive transfer of hypersensitivity. Use of fasting donors may reduce reactions due to food substances to which the recipient is sensitive. Such a practice would be of use in an exquisitely sensitive person,

but would be impractical on a large scale. Davenport[12] has eliminated all dietary rules for the donor and claimed no increase in allergic reactions was seen. Besides known substances such as food, other materials in blood may cause allergic reactions.[24, 48] These factors are usually manifest in repeated transfusions from the same individual when sufficient time has elapsed between transfusions to permit sensitization. Several cases are known in which the recipient developed a hypersensitivity by passive transfer; hypersensitivities have appeared to such materials as horse dander, horse serum, and strawberries following transfusion.[34, 57] Thus it would seem advisable not to use blood donated by a known allergic person.

Allergic type reactions have been attributed to other factors such as "early toxins" from nonpathogenic organisms or other enzymatic action in the blood.[26] In addition, the various principles which may cause pyrogenic reactions may be considered in this group since some have suggested an allergic basis for such reactions. This postulation is predicated primarily on the fact that both allergic and pyrogenic reactions are reduced by treatment with antihistamines.

Ferris, Alpert, and Coakley[17] have recommended the addition of antihistamines to blood (possibly also epinephrine hydrochloride) before transfusion in known allergic patients, with additional treatment by appropriate routes as required. Response was better when these drugs were used prophylactically rather than in treatment of reactions. The use of pyribenzamine hydrochloride in the blood reduced allergic reactions from 2.69 to 0.16 per cent and pyrogenic reactions from 4.31 to 0. per cent. Since 1,349 pints of blood were used, these reductions are significant. Hemolytic reaction symptoms were not masked in this investigation. The direct addition of procaine to blood before transfusion has been reported to reduce the incidence of pyrogenic reactions, but not allergic reactions.[46, 70]

Recently chlorprophenpyridamine maleate (Chlor-Trimeton Maleate) has received attention as an effective agent in reducing allergic and pyrogenic transfusion reactions. In a study covering several years' experience, Frankel and Weidner[20, 21] reported the incidence of allergic or pyrogenic reactions in control patients as 3.6 per cent (419 transfusions) while in those receiving blood containing the drug (10 mg. per pint) the incidence was 0.3 per cent (645 transfusions). Similar conclusions as to incidence reduction and dosage were reached by Simon and Eckman.[55]

Winter and Taplin[67] presented data on reaction rate reduction with Chlor-Trimeton in allergic and nonallergic patients. Some of the conclusions from this study were that allergic reactions occurred following transfusion about 10 times more often in known allergic persons than in nonallergic persons (febrile reactions are about equal); mixing the drug in the blood was more effective in reducing reactions than intravenous administration before transfusion; no serious drug reactions were noted, but mild drowsiness was experienced by most patients; small doses of the drug (2 to 4 mg.) given intravenously relieved the symptoms of immediate allergic reactions. These authors recommended the prophylactic use of antihistamines only in those patients having a history of allergic reactions. Winter and Taplin estimated that 1 of every 6 or 7 such patients has an allergic reaction in transfusion, while in nonallergic patients the rate is about 1 per cent. These investigators noted no effect on febrile reactions in contrast to other observers.[21, 55] The indiscriminate use of the drug in all transfusions is not recommended, especially in surgical procedures in which the patient is anesthetized and where multiple transfusions are given.

CONCLUSION

Several developments are reviewed which have reduced the incidence of transfusion reactions in recent years. Important among these are recognition of the Rh factor and other systems such as Kell, and the use of more sensitive methods in laboratory tests. However, the increasing use of plasma substitutes such as dextran may contribute. In regard to the use of dextran, the possible production of hypersensitivity should be considered in repeated administration.[30] Where only fractions of blood are needed, it seems advisable to utilize only that part essential to relieving the disease condition; for example, in anemia only the cells should be administered. Thus the possibility of introducing dangerous blood components is reduced. Barnard[3] has suggested a transfusion unit of human erythrocytes with cholinesterase and antibiotics. With dextran used in this preparation the cells are presumably maintained in such a state that they are less susceptible to hemolysis. Cholinesterase is added as a stimulant to reduce shock and to reduce red cell permeability. It is understood that many millions of pints of human cadaver blood have been used in Russia and Poland without serious reactions provided the blood was collected within six hours following death. The use of nonhuman blood products in transfusion is not promising.[60]

SUMMARY

Because of use of the exacting laboratory techniques available today and strict attention to the handling of each blood transfusion, reactions due to

the administration of incompatible blood occur infrequently. Allergic reactions are not predictable, but the incidence may be reduced by noting the past history of both the recipient and the donor. Possible causes of each type of reaction, their prevention and treatment are considered in the light of current knowledge.

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Surgical Treatment Of Cancer Of the Breast

MELVIN A. CASBERG, M. D.

Austin, Texas

IT IS the purpose of this paper to discuss briefly and simply certain basic principles in the surgical treatment of those stages of cancer of the breast in which radical surgery is indicated. The techniques of diagnosis are presented only insofar as these may relate to the major surgical procedure. The much debated question of preferential therapy for breast carcinoma does not lie within the purview of this presentation, other than the submission of certain clinical criteria of operability as outlined by Haagensen.[1] Carcinoma of the breast in women of all age groups who are in a condition to withstand surgery should be treated by radical mastectomy with these possible exceptions:

1. Extensive edema of the skin over the breast.
2. Inflammatory type of carcinoma.
3. Carcinoma developing during pregnancy or lactation.
4. Satellite nodules in the overlying skin of the breast.
5. Parasternal tumor nodules.
6. Edema of the arm.
7. Proved supraclavicular metastases.
8. Distant metastases.

Dr. Melvin A. Casberg, vice-president for medical affairs of The University of Texas, presented this paper at the Clinical Symposium on Management of Carcinoma of the Breast, The University of Texas M. D. Anderson Hospital and Tumor Institute, November 9, 1956.

The surgeon who treats cancer of the breast should apply his skills in psychological preparation of the patient and her husband and in rehabilitative measures postoperatively as well as in a gentle yet thorough surgical technique for best results. Both the patient and the surgeon should be ready to proceed with radical mastectomy immediately should the results of biopsy indicate its desirability.

The discerning surgeon, having evaluated the diagnostic pitfalls of chronic cystic disease with its menstrual rhythmic periodicity, must face the question of a space occupying lesion of the breast with only one answer, microscopic tissue examination. Parenthetically, there is only one place where this biopsy should be performed, and that is in the operating room, where all of the personnel and facilities are available for the completion of any procedure dictated by the findings of the frozen section. Aspiration biopsy is mentioned only for condemnation, for, though there are some authorities who advocate this technique, certain serious disadvantages such as the uncertainty of exact tissue source, the lack of clearly defined histology, and the repeated tissue trauma of the procedure preclude the use of this method in favor of incisional or excisional biopsy.

PREOPERATIVE PREPARATION

Preoperative preparation in anticipation of possible radical surgery involves a scope broader than is realized by many surgeons, ranging from the physical evaluation of the total patient to her psychological preparation for a disfiguring surgical procedure. Laboratory tests must clarify the body's general cellular and chemical fitness for the stress of trauma, and thereby aid in the determination of supportive measures to be applied before, during, and after surgery. Precaution dictates a minimum of a roentgenogram of the chest with further studies of the spine or other skeletal areas should the clinical history so suggest.

The role of the pathologist is a decisive one in this particular type of surgery, and he should be given every opportunity to familiarize himself with the various related factors. His careful examination of the patient preoperatively as well as his presence in the operating room during the biopsy will furnish supplemental information of aid in the microscopic diagnosis. Close cooperation in this manner accrues to the benefit of the patient.

Many doctors overlook the important role played by the husband in the psychological rehabilitation of the amputee. In younger and middle aged married women especially, there develops rather frequently a morbid sense of sexual imperfection following this type of surgery. The mental torture of this situation may far exceed the postoperative physical pain, and the surgeon has an obligation to do all within his power to prevent this complication. Careful indoctrination of the husband will help create an understanding and compassion which will tend to dispel mental anguish. In a similar manner, the surgeon should discuss with the patient such factors as the possible minimal limitation of shoulder motion and the availability of prosthetic appliances should the radical procedure be necessary. A patient thus prepared withstands both the psychological and physical trauma far better than one who faces these fears with ignorance and uncertainty.

OPERATIVE TECHNIQUE

Just as unsterile surgical techniques can spread bacterial contamination and infection, in like manner, careless handling of tumor tissue can disseminate cancer cells. Some surgeons unfortunately have associated radical surgery with a degree of roughness, incorporating stretching, tearing, and crushing into the procedure as though by such acts they were venting their wrath on the malignant process. No surgery demands a more careful and gentle handling of tissue than does that directed against the eradication of malignancy. Some years ago, the late George Crile divided surgeons into two classes: the rough or "carnivorous" type and the gentle or "herbivorous" type. The "carnivorous" surgeon has no place in the surgery for carcinoma of the breast.

BIOPSY

The patient is positioned and prepared on the operating table for radical surgery, including the preparation of the thighs as possible donor sites for skin grafts. Whenever possible, a general anesthetic should be used so that the patient does not suffer through the period of diagnostic indetermination. In the presence of the pathologist, the incisional biopsy is performed, removing the entire tumor if this is small or a section if it is large. Great caution must be exercised to prevent pressure or squeezing of the breast during the biopsy. While the pathologists report is awaited, preliminary hemostasis is carried out and the wound lightly packed with moist gauze. If the report is one of a benign lesion, the tumor, if it has not been removed, is excised and the wound closed hemostatically by careful apposition. If, however, the report is one of malignancy, the gauze may be left in the wound, the skin edges approximated carefully by a running suture, and the wound further sealed against the spillage of cancer cells by the application of collodion or some similar preparation.

At this stage, the entire operative area is considered to be contaminated and is re-prepared as in the original procedure, including a complete change of towels and drapes. The operating team participating in this primary act changes gowns and gloves and the instruments used are discarded as contaminated. There is no more logic to dispensing with these precautionary measures than there is to that of deliberately entering the peritoneal cavity through an abdominal wall carbuncle.

PRINCIPLES OF ADEQUATE SURGERY

Most operative techniques in radical mastectomy are modifications or adaptations of those developed by Halsted and Willy Meyer. In competent hands, the exact method makes little difference, as long as the exposure does not unnecessarily defunctionalize the shoulder and permits compliance with the four principles advocated by Halsted and Meyer, namely, excision of the skin overlying the tumor, excision of the pectoralis major and minor muscles, adequate axillary dissection, and removal of this entire tissue in one block. The step by step techniques of radical mastectomy are described in considerable detail by all standard textbooks of general surgery, and rather than repeat the minutiae of a more or less standardized procedure, it is the purpose of this paper to emphasize certain basic principles which are significant to the success of this therapeutic endeavor.

The first operative decision confronting the surgeon who accepts the responsibility for surgery of carcinoma of the breast is the adequacy of the procedure. If the patient's condition is such that she can be expected to stand a radical operation and if the spread of the malignant process appears to be limited to the area readily accessible to the scalpel, then any limiting compromise on the extent of the surgery is poor surgical judgment and contributes to a lower survival rate. Incomplete operations are still commonplace, due either to the surgeon's lack of confidence in the effectiveness of the radical attack or his lack of surgical competence. By way of clarification, adequate surgery as herein defined does not refer to the ultraradical methods involving such procedures as the partial osseous decortication of the thorax. It is difficult to associate the invasion of supraclavicular or mediastinal lymph glands with curability, and surgical exercises related to these areas still should be considered as experimental and of unproven value until further evidence is available.

SKIN INCISION

A typical skin incision giving adequate exposure for this procedure is one which, with the arm abducted to right angles, commences anterior and inferior to the tip of the acromion process over the cephalic vein which lies in the sulcus separating the deltoid from the pectoralis major muscle and then proceeds medially along this plane to a point inferior to the corocoid process, from where it is directed inferiorly and medially encircling the tumor area to terminate in the costo-xiphoid angle of the corresponding side. Under no circumstances should the incision be extended across or into the axilla, for the resulting scar may limit shoulder action. There has been and still is considerable discussion relative to the amount, both in depth and area, of skin which should be excised. Though one may plan to remove an area of skin extending at least 8 cm. from the palpable edges of the tumor, in the final analysis, good surgical judgment alone must dictate the limits of skin excision. Although it is true that experience will develop plans of general applicability, it is dangerous in all cases to regulate arbitrarily by fixed mathematical rules the skin area to be removed, for such a plan will result in excessive denudation in some patients and insufficient excision in others. Some surgeons pride themselves in procedures of a nature so extensive as to require routine major skin grafting. However, in a certain proportion of adequate radical mastectomies, the skin can be closed primarily and without tension; and again, obviously, there are those cases in which a procedure to be effective must leave denuded areas which will necessitate grafting. Certainly, the surgeon's approach to this decision should not be influenced primarily by the problems of skin closure, for the major question at hand is the eradication of cancer.

Inasmuch as the mammary gland embryologically is derived from the derma, one can expect a degree of structural and functional relationship between the breast and the overlying skin. Such an anatomical association dictates that, in the exposure of the operative area, a rather thin skin flap, including the dermal glands and a thin layer of areolar and fatty tissue, be dissected from the superficial fascia.

RESECTION IN CONTINUITY

If one were to attempt to develop a title describing the fundamental philosophy of the intent of radical mastectomy this might be epitomized in the words, "Noncontusive resection in continuity." Such a technique envisions the containment of the disease process within the limits of the tissue mass being resected in such a way that neither rough handling nor the surgeon's scalpel encroaches on the neoplasm and opens a Pandora's box of malignant cells to be disseminated into the highways of the lymph-vascular system.

That part of the surgical procedure related to the thoracic wall does not present any particular technical difficulties, for anatomical landmarks and cleavage planes afford rather constant and identifiable guidance. On the other hand, the axilla, with contents of a greater functional significance and of a wider range of positional variation, presents a distinct technical challenge. It is in this latter area that most of the inadequate and incomplete surgeries fall, and it is within the bounds of the axilla that effective surgery of necessity must be meticulous and time-consuming; otherwise, the term "radical" becomes a mockery.

Procedural planning of the thoracic portion of the operative steps, transecting the humeral, clavicular, and sternal muscle attachments and drawing the excised mass inferiorly and laterally, permits gravity to replace the tension of retractors, so that the weight of this musculo-glandular tissue itself provides a sufficient steady traction. This noncontusive technique of radical mastectomy has been described in detail by MacFee.[3] Removal of the anterior wall of the axilla thus exposes the axillary contents in such a manner as to permit a thorough dissection.

In dealing with cancer, all are agreed that one cannot compromise with the eradication of known accessible malignant cells; however, this truism should not be used as an excuse, in the presence of a lack of anatomical and physiological knowledge, for the unnecessary and wanton removal of normal tissue. Frequently, it is necessary to sacrifice blood vessels and even nerves in an effort to accomplish a cure. Macdonald[2] has shown that resection of the axillary vein not only is compatible with good function, but actually, in certain cases, reduces the secondary inflammatory processes which are conducive to the development of postmastectomy lymphedema. He concludes his presentation by stating, "resection of the axillary vein permits an adequate en-bloc dissection of the apical portion of the axilla."

From a technical standpoint, it is fortunate that the axillary vein and major venous pectoral tributaries lie anterior to the branches of the brachial plexus and axillary artery. The lymphatic system, lying in close relationship to the venous drainage of the anterior and lateral thoracic areas, is thus more readily available for excision. The subscapular, central, and subclavicular groups of lymph nodes are anatomically related to the axillary vein and its tributaries; thus, a meticulous dissection and stripping of the major vein as well as a ligation and excision of the venous branches are in order. As has been mentioned, if deemed necessary, sacrifice of the axillary vein may add to the thoroughness of the procedure. If possible, the thoracodorsal and long thoracic nerves should

be preserved, but even these should be removed if there is clinical evidence of involvement.

The entire surgical procedure should be carried out by sharp dissection, commencing superiorly along the cephalic vein, including the decortication of the axillary vein, and then continuing into the central axillary area with excision of the venous tributaries, lymphatics, and fatty areolar tissue as one mass. The forceful swabbing out of the axilla with gauze is a procedure to be condemned, for it not only fails to remove cancer cells, but actually may aid in their dissemination.

The gentle handling of tissues, careful hemostasis, elimination of dead spaces, and closure without tension are factors more directly related to a smooth postoperative course and the prevention of lymphedema than the actual extent of the surgical procedure itself. Skin closure must be accomplished without tension with the use of grafts when necessary. There are many techniques available[4] for the utilization of excess skin to close defects following radical surgery. Full thickness grafts from this source have an advantage in giving greater protection to the exposed thoracic cage.

The axillary dead space should be drained either passively by rubber drains or preferably by the use of active suction on one or more multiple perforation tubes distributed advantageously under the skin flaps. A pressure dressing of mechanic's waste or sponge not only will minimize oozing and stimulate adherence of the skin flaps, but also will contribute to the comfort of the patient.

POSTOPERATIVE CARE

Early ambulation and early use of the involved extremity will enhance convalescence, both physical and mental. The surgeon's duty is not completed with the discharge of his patient from the hospital, for he must continue in his program of rehabilitation and give close personal support until maximum function has returned. Periodic check-ups are essential, not only to observe and follow up on the effectiveness of the surgery itself, but also to give the patient the mental support and assurance necessary for her smooth convalescence from this particular type of psychic trauma.

CONCLUSION

In conclusion, it should be emphasized that factors contributing to the effective surgical treatment of cancer of the breast include the proper motivation on the part of the surgeon as well as the technical knowledge and facility to accomplish the procedure. The qualified surgeon who is convinced that the radical technique correctly applied affords the best chance for eradication of the malignancy is the surgeon more apt to succeed.

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Dr. Casberg, University of Texas, Austin.

Tumor Seminar

Conducted by

ARTHUR PURDY STOUT, M. D.

New York, New York

THE twelfth annual tumor seminar of the San Antonio Society of Pathologists was held at Brooke Army Hospital, Brooke Army Medical Center, Fort Sam Houston, Texas, October 29, 1955.

The meeting was opened by Col. Charles J. Farinacci, MC, commanding officer of the Fourth Army Area Medical Laboratory. Colonel Farinacci acknowledged the support given by the American Cancer Society and the South Central Region of the College of American Pathologists, and gave credit for the preparation of photographs to Dr. W. T. Hill of the Fourth Army Area Medical Laboratory and Henry Marasco of the Medical Illustration Section of Brooke Army Hospital.

Dr. Arthur Purdy Stout, professor of pathology at Columbia University College of Physicians and Surgeons, conducted this seminar for the San Antonio Society of Pathologists at Fort Sam Houston, October 29, 1955.

Colonel Farinacci then introduced Brig. Gen. Elbert DeCoursey, MC, commandant of the Army Medical Service School, honoring him as one of the prime backers of the annual tumor seminar, who together with Drs. B. F. Stout, A. O. Severance, J. M. Moore,

H. J. Schattenberg, S. W. Bohls, and D. A. Todd, comprised the original group that started the seminars in 1944.

(Dr. B. F. Stout, president of the San Antonio Society, was able to appear briefly during the session, when he gave credit for a great deal of the success of the annual seminars to Dr. Arthur Purdy Stout, who conducted the first two in 1944 and 1945, the proceedings of which were published in the *Texas State Journal of Medicine** and widely quoted.)

Major Gen. William E. Shambora, MC, commanding general of Brooke Army Medical Center, was introduced by General DeCoursey and expressed the pleasure of himself and the entire staff of the center in the presence of the group each year.

General DeCoursey introduced Dr. Arthur Purdy Stout of New York, who is professor of pathology at Columbia University and pathologist at the Institute of Cancer Research.

Dr. Stout: This is the fourth time that I have faced a similar audience under similar circumstances, and it has been interesting to me to observe in the intervening years the spread of popularity of sessions of this sort. I think that they have been really worth while. They have spread knowledge, and the present generation of pathologists is much better equipped to deal with tumor problems than they were in 1944. The differences that I notice in myself are less encouraging to me. I thought I was pretty good in 1944. I haven't looked it up, but I don't recall that I had much hesitancy about the diagnoses that I thought were correct for the different cases. Well, it is different with this set of cases that we are going to tackle today. I feel confident enough to make a definite diagnosis in two-thirds of the cases, but in one-third I have to make them with a question mark; I am far from sure that I am right in those. That is a saddening fact to me. I seem to have gone backwards in the intervening years, although I must say I've had an opportunity for a lot of experience in these years; but certainly I am not as good as I once was. However, perhaps we are growing new kinds of tumors, and the tumors are concealing themselves microscopically with various camouflages just to make it hard for us to recognize them.

CASE 1

Diagnosis.—Basal cell carcinoma (?) of scalp.

Contributor.—Col. C. J. Lind, MC, Brooke Army Hospital, Fort Sam Houston.

History.—A 20 year old boy had a progressive, slowly growing, nonulcerated lesion of the scalp for five years. Three months prior to biopsy it began to ulcerate superficially. The wound failed to heal. There were bilateral enlarged occipital nodes.

Submitted Diagnoses. — Adnexal (hair matrix and/or sweat gland) carcinoma, 21; squamous cell carcinoma, 12; basal cell carcinoma, 5; squamous cell and basal cell carcinoma, 2; adenocarcinoma, 1; undifferentiated carcinoma, 1; junctional nevus, 1.

Dr. Stout: I think everybody agreed that this was an epithelial type of growth, but it isn't generally agreed as to exactly what the nature of the growth was. It is interesting to me that one person even thought that it was a glandular carcinoma, and one thought it was a nevus or nevoid tumor. The diagnosis of junctional nevus surprises me a little because it seemed to me like a rather heroic form of junctional nevus.

I have four sections to study: two of them stained with hematoxylin and eosin and one each stained with Masson's trichrome and mucicarmine stains. The tumor, which lies chiefly in the corium but has invaded the subcutaneous fat and superficial fascia, is composed of freely anastomosing cords of spindle epithelial cells, the vast majority of which show no intercellular bridges and no keratinization or pearl formation. Unfortunately my sections show almost no areas where the epidermis and the tumor are both present. In one such place, two small groups of tumor cells show intercellular bridges and a tendency toward the formation of a pearl. Elsewhere the central parts of many of the thicker cords show central necrosis. The tumor infiltrates to a limited degree. The mucicarmine stain is negative for the tumor cells.

We must ask ourselves, is this a primary scalp tumor in a 20 year old boy which has been present ever since he was 15 years old, and if primary, what kind of a tumor is it? Its continued presence for five years without any suggestion of a growth elsewhere is strongly in favor of its being primary here in the scalp. If so, is it an epithelial tumor? The answer must be yes, not only on the basis of the appearance of the cells, but most of all because of the discovery of intercellular bridges in one group of tumor cells. This observation also automatically rules out the possibility of malignant melanoma. This leaves us with a choice between basal and squamous cell carcinoma coming either from the epidermis or some of the skin epithelial organs and structures. The major portion of this tumor has the general appearance and arrangement of a basal cell tumor, and the fact that in one group of tumor cells there is some epidermoid differentiation does not invalidate this point of view because such a variation is known to be of frequent occurrence.

Whatever this is, it is unusual because it developed in a 20 year old boy. It could be of course either basal or squamous. I could find, in the differential stains that I did, no evidence of secretion of mucicarminophilic material, and I would think that it was

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not any of the adenocarcinomatous types of growth with which I am familiar that come from the sweat glands or the sebaceous glands. I am inclined to rule those two out. I think that the diagnosis really lies between basal cell carcinoma and squamous cell carcinoma.

In my experience a squamous cell carcinoma of the skin, whether in young or elderly people, when it is completely undifferentiated is a truly malignant tumor and during a period of five years is apt to have metastasized. Moreover, the cases that I have seen have not grown in this fashion, which to me is much more suggestive of a basal cell type of growth. It is a really unusual tumor.

In my opinion, therefore, this is a basal cell carcinoma of the scalp which appeared first at age 15. This is, of course, unusual but not at all unknown. We are not told whether this boy has xeroderma pigmentosa or any familial tendency to develop such a tumor in childhood. However, I gather from MacKee and Cipollaro that isolated basal cell tumors have developed without known cause in children as young as 10 years. If this is accepted as a basal cell carcinoma, the chances are good that the enlarged occipital nodes on both sides are secondary to the ulceration of the tumor and are not due to metastases. The extreme rarity of metastases from basal cell carcinomas has been emphasized by Lattes and Kessler as well as by many others.

Now, I'll be glad to debate this with anyone who wants to debate it.

Dr. Béla Halpert, Houston: I beg to disagree on the nature of this tumor. Perhaps it is splitting hairs, but the naming of the neoplasm should provide information on one's thinking about its nature. In this particular instance the cells, to be sure, arose from cells of the epidermis. In the epidermis there is the basal layer of cells capable of division, and there are the cells nearby of the spinous layer also capable of cell division. We are accustomed to think that if the proliferation starts in the basal layer it will produce a basal cell tumor, and if it starts in the spinous layer it will be a squamous cell carcinoma. This concept is not valid. When a cell divides in the epidermis, it does not disclose characteristics of either of these cells. The direction in which the neoplastic cell differentiates depends on the stage of its development when it became a neoplastic cell. A basal cell tumor has properties of imitating a hair follicle, and it usually does this. I wouldn't make a diagnosis of any epithelial neoplasm which doesn't have a palisading cell layer on the outside of these cell nests and which sort of sweeps with the undetermined margin into, or touches, the supporting connective tissue. There is a fundamental difference between the basal cell tumors and the squamous cell carcinomas. The term "carcinoma" should not be used in connection with a basal cell tumor because that implies a stigma. A basal cell tumor is a borderline lesion between a benign and a malignant growth. I believe that all the morphologic evidence that is available here suggests that this is a squamous cell carcinoma rather than a basal cell tumor.

Dr. Stout: Well, there are two ways, of course, of approaching these tumors, one of which is the purely scientific way in which you attempt to sort out the tumors in order to give them the proper kind of name, depending upon the exact cellular origin, irrespective of the clinical behavior; whether or not, for example, the tumor will show progressive invasive characteristics and/or metastases. The ideal is to combine the scientific approach with the biological or clinical approach, because what we want to gain as aids to therapists is an insight into how a tumor is going to behave—what the chances of metastases are and what the chances of infiltrative growth and spread are. This tumor has existed already for five years and apparently it has not gone very deeply. It doesn't seem to me that an undifferentiated squamous cell carcinoma that had this degree of lack of differentiation would have remained as localized and without proved metastasis for all this length of time. I may be all wrong about it. I have also tried to be scientific, and I think there is a form of basal cell tumor that does not show the palisading that Dr. Halpert mentioned. At least I have used that term in some rodent ulcers, for example, that have gone on from the inner angle of the eye into the orbit, and eventually into the brain, behaving like the classical rodent ulcer, and they lacked any palisading. I am willing to admit that this lesion is more likely to belong to that class of growth than to the squamous class of growth.

Dr. J. J. Andujar, Fort Worth: I would like to point out that the picture which was projected was that of a Negro. Is this patient a Negro? And if so, would you comment on the incidence of basal cell carcinoma—I use the word "carcinoma" in its loose sense—in the Negro patient?

Dr. Stout: Well, I think it is much less common than in the white, and rather rare. I think that this is altogether an uncommon tumor anyway, and just on the basis of probabilities, I don't think we ought to place too much reliance on whether he is a Negro or not. You will go a long time, I think, before you see another tumor like this.

Col. C. J. Farinacci, Fort Sam Houston: This patient is a Negro.

Dr. Halpert: I want to ask Dr. Stout how he would have called this tumor if he hadn't had any history at all, just the slide?

Dr. Stout: I fluctuated around a lot when I first examined this, and it is practically impossible for me to answer that question because I did know the

history in advance; and how I would have thought about it, or what I finally would have thought about it, I really don't know. When I am prejudiced by the history, I can only guess what I might have said. I would like to be able to say I would have come to the same conclusion and think it is a basal cell carcinoma, but I really don't know that I would have done so.

Dr. F. C. Bornstein, El Paso: Since a question of the nature of basal cell carcinomas has been raised, do you consider them capable of metastasizing, or have you ever observed them metastasizing?

Dr. Stout: Yes, sir, I have. Dr. Lattes, who succeeded me in the job of surgical pathologist at the Presbyterian Hospital, reported 2 cases of metastasizing basal cell carcinoma: 1 that we had had at the Presbyterian Hospital and 1 that they had had at the New York Postgraduate Hospital. I think you will find, oh, a handful of authentic cases of basal cell carcinoma that would satisfy even Dr. Halpert as basal cell carcinomas, that have really metastasized; but it is so rare that I don't think one needs to take it into consideration in treatment. It is an oddity when one does metastasize.

Follow-Up.—At the time of the original operation (January 6, 1953) the left occipital lymph node was excised and histologically showed reactive hyperplasia. On September 1, 1954 (20 months later), the patient developed a mass in his right supraclavicular region, which was excised. It turned out to be tumor resembling the original scalp lesion. Chest roentgenograms, made in August, September, and December, 1954, and April, 1955, were essentially normal. Roentgenograms September, 1954, of the skull and mandible and lower dorsal vertebrae were negative, as was an intravenous pyelogram made the same month.

Gen. Elbert DeCoursey, Fort Sam Houston: There has been a request that Dr. Stout say something about "basal cell carcinoma" versus "basal celled carcinoma." I think this was brought up by Dr. Foot's article.

Dr. Stout: I don't think we ought to enter into etymological discussion at this time. It would carry us too far. I think, in spite of Chandler Foot, that probably it isn't quite as good English to say "basal celled" instead of "basal cell." But whether or not it is better English to say one rather than the other, they both intend and mean the same thing. The vast majority of people use the term "basal cell," and I don't think that Chandler Foot is going to be able to change that tendency. I have struggled in the past to put across other than the popular names for things and have had little success. I have had a struggle with most of the dictionaries of the country over the spelling of the word "neurilemoma," and the dictionaries are winning, as I felt sure they would, because they have an unfair advantage in that people tend to believe what they read in dictionaries. They are not controversial and curious enough to investigate to see whether what a dictionary says is really so. Usually it is, but not invariably. On several occasions I have had the strange task of reviewing medical dictionaries; I don't know how you would set about it, but it rather made me aghast to think of reviewing a dictionary. How could any one individual look over a dictionary and be qualified to comment, favorably or unfavorably, on all of the words in it? It is absolutely impossible. So I made up a way of doing it for myself, of simply looking up the words that I knew something about, and seeing how the dictionary handled them; and I regret to say that some of the dictionaries handled them very badly, indeed. I won't go any further into the matter, but don't believe everything you read in a dictionary, even as to spelling. It isn't always correct, or properly founded. Like every other thing the dictionaries tend to copy one another and former editions, without looking into the matter, whether or not the original dictionary word was justified or wasn't justified. So as far as I know, "basal cell" and "basal celled" mean the same thing, that is, a tumor presumably arising from the basal cells of the epidermis.

CASE 2

Diagnosis.—Malignant hemangioendothelioma of scalp.

Contributor.—Col. C. J. Lind, Brooke Army Hospital, Fort Sam Houston.

History.—A 63 year old white man had noted a progressive violaceous discoloration of the frontal scalp for the past six months. Following incidental trauma three months before, ulceration developed. The violaceous discoloration spread. There was no evidence of lymph node involvement. Following biopsy, the scalp was resected. The slide represents a section through the violaceous portion of the resected scalp. While the patient was in another hospital roentgen-ray films of lungs showed extensive metastases in both lung fields. The patient died three months postoperatively. Permission for autopsy was refused.

Submitted Diagnoses.—Malignant hemangioendothelioma (angiosarcoma), 23; hemangioendothelioma, 3; Kaposi's disease, 3; malignant melanoma, 5; amelanotic malignant melanoma, 2; basal cell carcinoma, 4; squamous cell carcinoma, 1; undifferentiated carcinoma, 1; hemangiopericytoma, 1.

Dr. Stout: The one favored, of course, is that form of angiosarcoma called hemangioendothelioma, with another variety of angiosarcoma, Kaposi's disease, coming in also; then a scattering of other diagnoses, even including hemangiopericytoma. Wherever I go to moderate seminars everybody thinks that there must be one case of hemangiopericytoma in the lot, and so in each case someone suggests hemangiopericytoma hoping to hit the bull's-eye.

The characteristics of this fascinating tumor are that it seems to be made up of many freely anastomosing dilated capillaries which are lined by anaplastic cells showing mitoses. In places the tumor

cells form solid cords without lumens and in still others tumor cells form solid amorphous masses. A silver reticulin stain shows that the tumor cells do not form connective tissue fibers of any kind. The Masson trichrome stain confirms this, and the mucicarmine stain shows no evidence of secretion by any of the tumor cells. In the more solid portions of this neoplasm it is possible to observe that there are also unaffected blood vessels scattered among the undifferentiated tumor cells.

This is an example of a rare malignant tumor. There is little doubt in my mind that this is a malignant hemangioendothelioma. There is no reason to suppose it is a metastasis from a chorionepithelioma, which is the principal mistake in diagnosis sometimes made because it can imitate this tumor; but the tumor cells in this growth are uniformly the same and there are no evidences of syncytia formation. It seems safe to exclude any of the malignant epithelial tumors which might be primary here or might be metastatic. When I looked up our cases of malignant mesenchymal tumors in September, 1951, out of 1,349 only 34 were classified as malignant hemangioendothelioma. I was unable to find a suitable case of this kind during a two-year period when I was preparing the cases for the Seminar on Tumors of the Soft Tissues for the American Society of Clinical Pathologists.

As of September 1, 1955, this figure has nearly doubled, for there are now 65 cases of malignant hemangioendothelioma recorded in our laboratory. There are a few doubtful cases in addition, which I have omitted. The neoplasms have quite a wide distribution throughout the body tissues, as table 1 will show.

TABLE 1.—*Malignant Hemangioendotheliomas Recorded in Surgical Pathology Laboratory, Columbia University College of Physicians and Surgeons Up to September 1, 1955.*

Tissues Other Than Soft Tissues		Soft Tissues	
Bones	10	Lower extremities	5
Breast	10	Upper extremities	3
Spleen	6	Muscles	3
Liver	2	Neck	5
Penis	2	Head	3
Pleura	1	Trunk	5
Sciatic nerve	1	Retroperitoneum	2
Nasal cavity	1	Mediastinum	1
Pharynx	1	Orbit	1
Pancreas	1	Joint	1
Uterus	1		
	36		29

This is still a very uncommon malignant tumor. It varies a good deal in malignancy. In the breast it is almost always fatal and is found more often in adolescent girls than in adult women. In the bones it is variable in its behavior and does not always metastasize with any speed. Several of our cases have involved several bones. The small number of cases seen in any one area makes it extremely difficult to predict the behavior with assurance. Almost all cases are characterized by a free interanastomosis of tumor vessels. I do not like to make the diagnosis unless I can find somewhere evidence of multiplication of endothelioblasts in freely anastomosing vessels. Other parts of the tumor may show only sheets of tumor cells without evidence of formation of reticulin fibers, but unless I can find proof that the tumor cells are derived from cells lining capillaries I am unwilling to make the diagnosis.

Now, this to me was definitely a malignant vascular tumor—a rare one, in which the dominant cell type is the endothelial cell. In my classification you can put all the malignant vascular tumors under the general heading of angiosarcoma, if you wish, and then divide that up into subheadings such as the hemangioendothelioma, in which the endothelial cell is the dominant one; the hemangiopericytoma, in which the pericyte is the dominant cell; Kaposi's sarcoma, that highly specialized growth which some people perhaps would like to remove from the vascular category and put into the malignant lymphoma class because some of them are associated with either Hodgkin's disease or lymphosarcoma, but to me that is fantastic. I cannot ever bring myself, I am sure, to call Kaposi's sarcoma a malignant lymphoma. The fourth variant is the lymphangiosarcoma that develops in the swollen arm after roentgen-ray treatment of cancer of the breast, and that can occur even in other places. If you want to add a fifth group, there are the very rare malignant lymphangiopericytomas.

Dr. Béla Halpert, Houston: I want to compliment Dr. Stout on pronouncing Kaposi correctly. That is a rare occurrence.

Dr. Stout: I was taught how to pronounce it by a Hungarian. I don't say Ka-pō'-si's.

Dr. Halpert: Is there any difference between hemangioendothelioma and Kaposi's disease, the lesion that Kaposi called hemorrhagic sarcoma? I agree that this is a hemangioendothelioma, compatible with Kaposi's angiosarcoma.

Dr. Stout: In my opinion they are different. The picture of Kaposi's sarcoma that I use in diagnosis is a tumor in which there is a rich capillary network set in a stroma featuring cells that look like fibrosarcoma cells. I don't see any of that featured here. That is the characteristic picture; of course there are variants from it, but in the great majority of instances that is the picture. I have had some correspondence with Dr. J. F. Murray of the South African Institute for Medical Research in Johannesburg, South Africa, where, as you may know, Kaposi's sarcoma is so much more common in the Bantu colored people than in whites, whereas in this country the reverse is true.

In fact, we exchanged slides on a number of cases because he wanted to see whether the lesions we were calling Kaposi's sarcoma in this country were reproduced in exactly the same way in the Bantu, and they were. There was no difference, histologically. But to me the clinical picture in this particular case is different. I would think that it would be at least unlikely, or rather rare, that Kaposi's sarcoma would make one great big spreading lesion in the skin and subcutaneous tissue without appearing any place else except in a definite metastatic fashion, and I would also think it was extraordinarily rare that it should kill a person in nine months. I've never known classical Kaposi's sarcoma to move that fast. Maybe it does, but to me that is unknown.

Dr. Halpert: I use another criterion, too, in diagnosing Kaposi's disease, namely, the presence of a little bit of fibrin along the vascular spaces, but that feature was not prominent in this case.

Dr. A. O. Severance, San Antonio: Dr. Stout, in the years that have gone by I have come across tumors that I thought were hemangioendotheliomas in which the proliferating cell was within the reticulum sheath, and in which the cells were fairly uniform in size and shape and much smaller than these here, with frequent mitotic figures. I've called those hemangioendotheliomas and thought that they were of the benign type. They occurred commonly in children, but sometimes in adults, and they behaved in a somewhat benign fashion. I wonder if you would take this opportunity to comment on that, whether you think there is a benign type.

Dr. Stout: Indeed I do, and in order to make certain that there shall be no confusion in the clinicians' minds if we use the word "hemangioendothelioma," I always preface it by either "benign" or "malignant" so as to make it perfectly certain that if we make a diagnosis of hemangioendothelioma and it is one of the benign varieties you have described, it will not be mistaken for one of the malignant tumors. I think one of the distinguishing features, certainly, is the character of the cells. In the benign type the cells do not look like anaplastic malignant tumor cells. In the malignant type they do, very definitely.

Gen. Elbert DeCoursey, Fort Sam Houston: How is the reaction to radiation in these tumors, Dr. Stout?

Dr. Stout: I never knew anything to stop the malignant hemangioendothelioma. You may kill off a few cells, but I never knew any case to be cured by any means at all, surgery or irradiation, in these truly malignant hemangioendotheliomas. I think they are terrible and vicious tumors. I do not recall any one of them in which radiotherapy destroyed even part of the tumor, and my impression is that they are radioresistant as well as incurable by radiotherapy.

CASE 3

Diagnosis.—Rhabdomyosarcoma of buccal mucosa.

Contributor.—Capt. R. C. Schaffer, USAF Hospital, Lackland Air Force Base.

History.—A 22 year old white man had a tumor of the left buccal mucosa which was rapidly enlarging. It was incised, and a diagnosis of malignant tumor was made. On trans-shipment to another base the patient suddenly died. Tumor masses were found involving other sites such as pancreas, pleura, lungs, heart, thyroid, liver, and adrenal glands.

Dr. Stout: We made some additional inquiries. The tumor in the cheek was regarded as the primary site. The onset of illness was in April, 1954, and death occurred June 16, 1954, so this again is a rapidly growing tumor. The left submaxillary and submental lymph nodes were said to have been involved grossly at autopsy, but there is no mention of them microscopically. The metastases mentioned were all in organs. I wanted to find out whether there was any lymph node involvement, and that is the reason for that statement.

Submitted Diagnoses.—Rhabdomyosarcoma, 18; reticulum cell sarcoma, 5; fibrosarcoma, 4; hemangioendothelioma, 3; hemangiopericytoma, 2; bronchogenic carcinoma (anaplastic), 2; miscellaneous malignant tumors, 7; "eeny meeny miney mo," 2.

Dr. Stout: It doesn't seem to me that we got as many people making diagnoses in this case, but the majority evidently thought it was a rhabdomyosarcoma; and there is hemangiopericytoma raising its ugly head again. Obviously somebody here thought that it was a metastatic lesion in the cheek. You can interpret that last diagnosis any way you want. How did two people think of that? There must have been collusion there.

This tumor is composed largely of rounded, undifferentiated cells. Scattered among them are varying numbers of extremely long strap cells and occasionally large rounded cells. In the hematoxylin-eosin section the cytoplasm of these last two cell varieties does not differ much from that of the undifferentiated cells, but with Masson's trichrome stain the cytoplasm is a brilliant red while the undifferentiated cells remain a neutral tint. Even though one cannot find cross striations, there are longitudinal fibers and in the large rounded cells a division of the cytoplasm characteristic of embryonal striated muscle cells. No normal striated muscle is identified.

The presence of these long strap cells and the large rounded cells with acidophil cytoplasm is enough to make the diagnosis if one is satisfied that they are tumor cells and not separated remnants of invaded striated muscle. I am satisfied that in this case they are neoplastic and that the tumor belongs to the group of rhabdomyosarcomas. I would like to point out that this is more like a juvenile rhabdomyosarcoma than cases more commonly seen in adults. In

the latter it is seldom possible to find sufficiently differentiated tumors to make them easily recognizable. One often has to depend upon bizarre multinucleated giant cells, some of which have peripherally arranged vacuoles containing glycogen—the spider-web cells. It is much easier to call a tumor a rhabdomyosarcoma in an adult and be in error than is the case with children. I would stress particularly the fact that there is a giant cell carcinoma in the lung which can imitate a rhabdomyosarcoma; and there can occur in the upper respiratory and alimentary tracts projecting growths suggesting rhabdomyosarcoma which may develop either spontaneously or following radiotherapy and often may be associated with intramucosal or infiltrating squamous carcinoma at the base. These giant cell tumors are usually not even neoplasms. I wish I knew some way in which one can surely recognize these pseudo tumors. One simply must know where they are apt to develop and be aware of them so as to avoid wrong diagnoses leading to unnecessary mutilating operations.

When I wrote the fascicle on tumors of the soft tissues and dealt with the subject of rhabdomyosarcoma, I see that I did not devote enough space to the discussion of the difference between juvenile and adult rhabdomyosarcoma, nor to the difficulty of distinguishing the pseudo from the real rhabdomyosarcoma. Let this discussion be a supplement to the fascicle.

There are three varieties of rhabdomyosarcoma in children. The first is represented by the tumor type known as sarcoma botryoides, found most frequently in the pelvic genitourinary tract of males and females and characterized by the formation of grapelike myxoid nodules with stellate rhabdomyoblasts sometimes showing cross striations. There are also small rounded rhabdomyoblasts, sometimes but not always showing strongly acidophil cytoplasm. The second variety of juvenile rhabdomyosarcoma found in the retroperitoneum, the nares, orbit, external canal, and middle ear, and occasionally in muscles elsewhere, is featured especially by the small rounded cell previously described and less often by myxoid areas with stellate cells. Both varieties may show the long strap form and the large rounded forms found in this tumor. The third variety is found especially in the heart. This is characterized by large bizarre rhabdomyoblasts often showing cross striations. Some prefer to call these heart tumors rhabdomyomas, believing that they are congenital malformations and benign. Finally, rhabdomyosarcoma is not infrequently one element in a mesenchymoma or in such a teratomatous growth as Wilms's tumor of the kidney. In the adult, as already pointed out, the juvenile small rounded and stellate rhabdomyoblasts are usually not seen and one has to depend upon bizarre giant cells and spider-web cells. The search for cross striations may be very onerous or fail entirely.

Dr. Béla Halpert, Houston: Were these striations discernible in the hematoxylin-eosin sections that we saw? And does it require special fixing fluid such as absolute alcohol to demonstrate glycogen?

Dr. Stout: If one has the patience to hunt long enough, I think cross striations can be found in the hematoxylin-eosin stains. I think the special stains facilitate finding them, because it makes them more easily apparent; but one can see them if there is a reasonably good hematoxylin-eosin preparation. It's got to be reasonably good, though; otherwise you will not see them.

Can one demonstrate glycogen without special fixation? Well, if you are lucky, you can, even after formalin or Bouin's, and you don't always have to have the special fixative. Of course you will get it much more reliably if you do have the alcohol.

Dr. Halpert: I was asking that question because I had a case of glycogen-storage disease in which the autopsy was performed 60 hours after death. The spaces were there in all the organs, particularly in the muscle, myocardium, the kidneys, and the liver; but I couldn't demonstrate the glycogen, which was gone. The article wasn't accepted for publication by Dr. Hektoen because I couldn't actually demonstrate the glycogen, although I knew it was there.

Dr. Stout: Well, that was high intellectual and scientific integrity on the part of Dr. Hektoen.

CASE 4

Diagnosis.—Fibrosarcoma (?) or mesenchymoma (?) of breast.

Contributor.—Dr. Sylvia Johns, Nix Laboratories, San Antonio.

History.—A 79 year old white woman first noted a lump in her breast one week prior to consulting her doctor. There had been no trauma, no hormone therapy, and no nipple discharge. There was no history of cancer, diabetes, or tuberculosis in the immediate family. The patient had nursed 10 children. At physical examination she appeared well. There was a hard, irregular, 5 cm. tumor in the upper outer quadrant of her right breast. There was a positive plateau test, poor transillumination, and nipple retraction. Chest film was negative for metastasis. The specimen consisted of an unencapsulated fatty mass of incised breast tissue measuring 7 by 6 by 6 cm. with a central tumor measuring 4.5 by 4 by 4 cm. This tumor was circumscribed for approximately one-half of its circumference but no definite capsule was made out. The tumor showed extensive necrosis and cavitation with the periphery soft, moist, and gray-pink with foci of necrosis and hemorrhage. Peripheral to the tumor were multiple dilated ducts with creamy secretion. Excision biopsy with frozen section was followed immediately by simple mastectomy with excision of one axillary node. The patient had a full course of roentgen-ray therapy. She noted recurrent lumps in the region of the scar within one month and died within six months. No autopsy was performed.

Submitted Diagnoses.—Breast carcinoma, 12; fibrosarcoma, 10; liposarcoma, 5; osteogenic sarcoma, 1; hemangio-

pericytoma, 2; carcinosarcoma, 4; cystosarcoma phylloides, 2; miscellaneous malignant tumors (sarcoma; carcinoma), 7.

Dr. Stout: I expected we would get various diagnoses. There it is—[hemangiopericytoma]! Well, keep trying, you might hit it! Cystosarcoma phylloides—Colonel Farinacci, I recommend that your secretary look up the spelling of phyllodes. I should like to know if there is a dictionary that spells it that way.

Col. Charles J. Farinacci, Fort Sam Houston: Dr. Stout, I had this out with my secretary, and she promptly brought out her twenty-second edition of Dorland's "Medical Dictionary," 1951, and showed me that this term, as it is spelled here, is preferred. In parentheses there was the other one without the "i," and I said "Well, I guess I am licked, but it isn't right."

Dr. Stout: Well, for me, that is a wrong spelling.*

This tumor appears to be a sarcoma without any evidence of an epithelial element. It is made up largely of spindle-shaped cells arranged pretty much haphazardly. Only a small portion of the section shows viable cells; the rest of it is necrotic. One can see that there are capillaries coursing in various directions in the viable tumor tissue; elsewhere there are very few capillaries. Trichrome and especially Laidlaw silver reticulin stains show many reticulin and some collagen fibers between and around a majority of the cells. A few groups of amorphous tumor cells have no fibers about them. Mitoses average about one in every three high power fields. The mucicarmine stain shows no intracellular red or pink secretion vacuoles.

We still have a great deal to learn about the malignant noncarcinomatous tumors of the breast. I have made two attempts to set up criteria for naming them and for predicting the result following various types of treatment and have tried to correlate our results with cases reported in the literature. In the case of cystosarcoma phyllodes it is possible to recognize a majority of them, but we have not learned how to predict the result of treatment or the biological behavior if untreated with any assurance at all. When it comes to the sarcomas not developing in adenofibromas of the breast, we have recognized fibrosarcoma, liposarcoma, rhabdomyosarcoma, osteogenic sarcoma, malignant hemangioendothelioma, and malignant mesenchymoma. The record of the microscopic appearance and the results of treatment of our cases can be found in the paper by Lester and Stout. The number of cases is few aside from cystosarcoma phyllodes, and the behavior of the anaplastic tumors following simple mastectomy unpredictable; for this sometimes cured for a long period of years but sometimes metastasis through the blood stream led to rapid death, as in this case. Malignant hemangioendothelioma primary in the breast was an exception, for it was always fatal and in most cases with extreme rapidity. Rhabdomyosarcoma was the only type which metastasized to the lymph nodes in the axilla.

What shall this tumor be called? As in many other cases we are asked to judge from a small fragment of a 5 cm. tumor much of which is necrotic. I am always fearful that sections from some other part of this tumor may show some other kind of differentiation. If they did so, the tumor would be a malignant mesenchymoma. If this small fragment is representative of the entire tumor, I am forced to call it a poorly differentiated fibrosarcoma. The tumor cells are largely surrounded by connective tissue fibers which seemingly they have formed. The fact that small groups of cells are not surrounded by fibers does not invalidate the diagnosis of undifferentiated fibrosarcoma; but in cases of apparent undifferentiated fibrosarcoma one must remember always that some or all of the cells might be of some other type and could have assumed the functions of fibroblasts. Without any proof that this tumor is other than a fibroblastic one, I can see no escape from calling it a malignant fibrosarcoma. The number of mitoses is sufficient to necessitate calling it malignant, although it would not have been possible to have predicted the extremely rapid course.

Dr. Béla Halpert, Houston: I don't recall whether or not I called this a liposarcoma, but I probably did. In liposarcomas at times the connective tissue element looks like a fibrosarcoma, and even the larger part of the tumor may be composed of that element. I would like to comment on neoplasms of nonepithelial origin in the mammary gland. It is strange that this organ which has so much adipose tissue and so much connective tissue in it is so rarely the site of either a pure fibroma or a lipoma. The only fibroma of the mammary gland reported in the last 25 years was seen by me.*

Dr. Stout: Oh, I think that is perfectly true, and it isn't limited to the mammary gland. The whole distribution of lipomas is peculiar, and their behavior is peculiar, too. I think we must have half a dozen lipomas of the mammary gland, and in my observation they occur in two different ways: In one, the lipoma is in the mammary gland, but has no mammary glandular elements in it at all. It is separated from them by a capsule. It may not be a perfectly rounded tumor, but it is separated from all of the mammary elements. In the other, the fat is evidently infiltrating, because the mammary acini or ducts are found scattered through it. That is of interest to us

Stedman's "Dictionary," eighteenth edition, 1953, says the Greek is "phyllodes" meaning "like leaves." Blakiston's "New Gould Medical Dictionary," 1949, gives "phylloides" but does not give the Greek justification for this spelling.

*Halpert, B., and Dial, D. L.: Fibroma of Breast, Am. J. Path. 9:905-908 (Nov.) 1933.

pathologists, but as far as I know the fatty tumors in the breast that justify the term lipoma are always benign and, as far as my experience goes, have never given rise to a malignant liposarcoma.*

Dr. Leo Lowbeer, Tulsa, Okla.: Isn't the biological behavior of this particular neoplasm unusual? Would you have predicted after seeing the slides that the patient would die so rapidly? I remember that we had a case of malignant mesenchymoma of the breast which Dr. Stout discussed in a seminar in Tulsa. This tumor had a far wilder morphologic aspect. It had a tremendous number of mitotic figures. It was far more cellular. Yet the patient survived for five years. Nobody would have predicted that. Could you make some comment on this?

Dr. Stout: Yes, I agree that morphology is no sure guide to biological behavior, and the more one encounters tumors in different parts of the body, the more he will become convinced of that. You only have to think of the relatively benign appearance of thyroid carcinomas and their malignant behavior, and the same statement applies to occasional tumors in all parts of the body. One cannot give any kind of a guarantee as to what the behavior of a given tumor will be. All one can say is that the majority of tumors of this or that type have statistically behaved in this or that fashion; but you will always encounter, if you live long enough, an exception to the rule. And I have lived almost long enough to encounter exceptions to all rules.

Dr. D. Useda, San Antonio: I think I read that when a tumor has a fibrosarcoma pattern and you find tumor giant cells, that tumor is not called a fibrosarcoma, but you should think of a liposarcoma or rhabdomyosarcoma. I wish Dr. Stout would make some comments about that.

Dr. Stout: I don't know whether I said that in print, but I certainly agree with that completely. I think the true, simple fibrosarcoma seldom produces any appreciable number of giant cells, so that whenever I see a fibrous tumor that has any considerable number of giant cells in it, I always look about to see if I can't detect some other features which will enable me to put it in its proper class—lipo-, rhabdo-, or even some other kind of sarcoma.

CASE 5

Diagnosis.—Rhabdomyosarcoma (sarcoma botryoides) of the common bile duct.

Contributor.—Col. C. J. Lind, Brooke Army Hospital, Fort Sam Houston.

History.—A 6 year old boy developed dark urine, jaundice, pruritus, and clay colored stools within five days. Nineteen days later, on hospital admission, there was a temperature of 104 F., jaundice, and nontender liver enlargement of two fingerbreadths. White blood cell count was 17,500 with 77 per cent polymorphonuclear leukocytes and 23 per cent lymphocytes. Hemoglobin was 10 Gm., red blood cell count 3,200,000, and sedimentation rate 36. Heterophil, cold and febrile agglutination, Coombs' test, and blood culture studies were negative. A gastrointestinal series showed an extrinsic pressure defect of the second portion of the duodenum. An exploratory laparotomy revealed a 1.8 by 2.7 by 3 cm. tumor mass with peripheral grapelike fragments obstructing the distal portion of the common duct. One section was taken through the main tumor mass.

Follow-Up.—Postoperatively the child's condition deteriorated rapidly, and he died April 17, 1955, approximately nine weeks after initial onset of symptoms. The autopsy findings showed the presence of the original tumor of the common bile duct with extension into the head of the pancreas and wall of the duodenum. In addition there were periductal lymph node metastases.

Submitted Diagnoses. — Sarcoma botryoides of the common bile duct, 17; leiomyosarcoma, 6; rhabdomyosarcoma, 5; fibrosarcoma, 3; liposarcoma, 2; mesenchymoma, 3; neurogenic sarcoma, 1; myxosarcoma, 1; foreign-body granuloma (cotton), 1; miscellaneous diagnoses, 4.

Dr. Stout: I suspect that a number of people probably read *Cancer* and saw Dr. Horn's case published there, while the rest did not. We have a good scattering of diagnoses, practically all except one of them indicating that the diagnostician thought that the tumor was malignant; and we don't know what the miscellaneous diagnoses were.

Now let's have the roentgenograms, and I will ask Col. Henry C. Harrell, MC, radiologist at Brooke Army Hospital, if he will interpret the film.

Colonel Harrell: There is an area of obstruction to the second portion of the duodenum. This was a constant finding and not just a peristaltic wave. The duodenal bulb is also considerably enlarged, and the stomach is fairly large. In order to tell the emptying time of the stomach, of course, additional films would have to be made. This obstruction is extramural. The mucosa inside the duodenum appears to be fairly well intact, and certainly adjacent mucosa is intact. One of the causes of obstruction at this point, of course, is a tumor or tumefaction in the head of the pancreas; but the common bile duct comes right along here, and in this particular case undoubtedly the enlargement of the common bile duct at this point causes this obstruction. It is possible that there could be some invasion of the wall. An intramural tumor could cause this obstruction, but one would expect a little more disturbance of the mucosa lining the duodenum at that point.

Dr. Stout: Thank you, Colonel Harrell.

Where this tumor invades and forms projecting masses in the mucosa of the common duct, it is largely myxoid with the intercellular substance stained pink by mucicarmine and with most of the cells appearing stellate as a rule, but with some areas where

As of February 6, 1956, there are records of 88 lipomas of the female and 2 of the male mammary gland in the Surgical Pathology Laboratory of Columbia University.

there are small rounded undifferentiated cells. As soon, however, as one gets away from the polypoid surface formations and into the solid underlying tumor, one finds a new cell form, the long strap-shaped cell, in great numbers. The hematoxylin-eosin preparation shows that the cytoplasm of these cells is acidophil; but a really striking picture is obtained by using Masson's trichrome stain, when it is at once apparent that the cytoplasm is intensely red and one recognizes these cells as strap-shaped rhabdomyoblasts. Cross striations can be found in both stains. They are not very frequent. I think that we can accept this as a definite rhabdomyosarcoma of juvenile type, and of the special type known as sarcoma botryoides.

Recently a case similar to this one was reported by Horn and his associates. Dr. Horn had showed his case to me. Of course I agreed with his diagnosis and was astounded at seeing sarcoma botryoides in the common duct of a child. Horn painstakingly combed the literature when reporting his case and found recorded 2 previous common duct cases which grossly and possibly microscopically imitated this appearance. Neither of them had been recognized as rhabdomyosarcomas by their reporters, but Horn thought that that is what they were. At my request Dr. Horn included his case in the September, 1954, seminar of the Arthur Purdy Stout Club in Washington, D. C., so that it was easy for Colonel Farinacci to diagnose this present case when it came his way. Since Dr. Horn's case Colonel Farinacci has had the good fortune to see 2 cases that reproduced the same picture and were recognized by him as sarcoma botryoides of the common bile duct.

I have spent some time in discussing the histological characteristics of juvenile rhabdomyosarcoma when dealing with case 3. It seems unnecessary to repeat what was said there, but I might enlarge on that by a couple of other remarks. I used to think that sarcoma botryoides sometimes was a pure myxoma, and because in those cases I didn't succeed in finding any cross striations, I didn't think that they were probably definite rhabdomyosarcomas. I have changed my mind now. I believe that all cases of sarcoma botryoides are rhabdomyosarcomas of a peculiar type. Perhaps the fact of projection into a cavity or duct or on the surface of the mucous membrane is responsible for the myxoid appearance, but I believe all those stellate cells are really rhabdomyoblasts although they may not have differentiating features. Whether or not there is such a thing as a pure myxoma of the genital sphere I do not know.

I cannot believe sarcoma botryoides of the common bile duct will ever be a common tumor, and this audience may never see another case. Why juvenile rhabdomyosarcomas develop where they have been found in rather restricted regions of the body is an observed fact which so far has defied interpretation.

Let's ask if anybody has seen a case like this of sarcoma botryoides of the common duct. We will let it go on record that nobody has.

CASE 6

Diagnosis.—Granular cell myoblastoma of anterior neck region.

Contributor.—Dr. A. O. Severance, Baptist Memorial Hospital, San Antonio.

History.—A 17 year old white girl had a six month history of enlargement of what was thought to be the right lobe of the thyroid. At surgery after approximately two-thirds of the right lobe of the thyroid had been removed, a tumor was discovered posteriorly beneath the thyroid. This was shaved from the trachea. The gross specimen was a 7 Gm., smooth, ovoid mass, 3.5 by 2.5 by 1.5 cm. The cut surface showed a homogeneous, firm, yellowish white tissue with a fibrillar structure.

Follow-Up.—When the patient was seen approximately 11 months after surgery, she was well and there was no recurrence.

Dr. Stout: I might remark that when this history came to me it said that the tumor lay below the thyroid. Well, "below" unfortunately might be interpreted differently whether the patient was lying down on her back or whether she was standing, because in one position it would be posterior to it, and in the other position it would be inferior, or caudad to it. I learned from Dr. Severance that this one was posterior to the thyroid. The nodule was adjacent to the trachea, more on the right side, and adherent to it.

Submitted Diagnoses.—Granular cell myoblastoma, 33; parathyroid adenoma, 3; hibernoma, 1; Hürthle cell carcinoma, 1; nonchromaffin paraganglioma, 2; alveolar soft part sarcoma, 1; rhabdomyoma, 2.

Dr. Stout: As I expected, almost everybody said that it was a granular cell myoblastoma. We've got a little scattering of differences of opinion; and the diagnosis of alveolar soft part sarcoma would be a synonym for malignant granular cell myoblastoma. The same remark applies to nonchromaffin paraganglioma, so that three people believed that this was a malignant tumor, and the majority, I take it, did not.

This tumor is made up of cords of plump, elongated, and sometimes almost rounded cells of relatively large size with small rounded nuclei and a voluminous cytoplasm filled with fine, moderately acidophil granules. The Laidlaw reticulin stain shows that the cords of cells do not have reticulin fibers between them, but one cord is separated from its neighbor by a dense tangle of fine delicate reticulin fibers. Intermingled with the tumor cords are widely separated striated muscle fibers. No nerve twigs are detected in these slides.

Obviously this is a granular cell myoblastoma of

the ordinary type which has no suggestion of malignancy. That this variety can become malignant I can testify, for I have seen 5 cases in which the primary tumor showed no recognizable evidence of malignancy, except perhaps that some of the nuclei were a little larger, but which nevertheless metastasized. When I prepared the discussion of benign granular cell myoblastoma for the fascicle on tumors of the soft tissues, there were 120 cases recorded in our laboratory. This was not including the malignant variants of which we had 4 of the simple kind and 12 of the organoid granular cell myoblastomas. Four years later as of September 1, 1955, 60 benign cases have been added, making a total of 180. In this later group of 60 cases the tongue still leads all other sites, with 8 cases. The situation of this present case is unique so far as I am concerned. We have none either in or near the thyroid gland and only 1 case in the neck at all. It was apparently subcutaneous in the lateral neck.

There is nothing new to report concerning the cellular origin of the granular cell myoblastoma. At least one set of these tumors apparently does not arise from myoblasts; these are the pedunculated tumors attached to the gums of newborn infants. The cells composing them look just like the granular cell myoblast except perhaps they are more apt to be rounded. However, since cells of this type can appear occasionally in adamantinomas I am content to believe that growths on the gums of newborn infants are probably malformation tumors of the embryonal enamel organ. As for the malignant organoid myoblastomas, I am still waiting for proof that they are derived from nonchromaffin paraganglions, because of Murray's observations in tissue culture which showed no difference in cellular growth pattern in vitro between ordinary and organoid granular cell myoblastoma. I keep an open mind, but I see no reason to change the name without convincing evidence.

Dr. Leo Lowbeer, Tulsa, Okla.: Could you, Dr. Stout, comment on the validity of the argument that these tumors could not originate from striated muscle cells since they are so frequently found in locations where there are no striated muscles? We have seen a tumor of this sort in the bronchus, where there was some reason to assume that the tumor may have originated from smooth muscle cells.

Dr. Stout: I can't make any intelligent comment on that except to say that tumors that we will all accept as rhabdomyosarcomas because they develop with cross striations and other features can appear in parts of the body where there are no known rhabdomyoblasts; so that isn't, to me, a bar to their being rhabdomyoblasts. I don't say that they are; I don't say that they are not.

Dr. J. L. Goforth, Dallas: Isn't it rather confusing, Dr. Stout, to refer to a benign-behaving tumor as a myoblastic tumor?

Dr. Stout: To me, using the suffix "blast" simply means that the lesion is reproducing in its growth an embryonal type of cell; that embryonal type of cell doesn't necessarily connote malignancy. The rhabdomyomas of the heart, for example, are not in any sense of the word made up of adult types of heart muscle cells. They are composed of bizarre, embryonal type of heart muscle cells distended with glycogen, yet they are nonmetastasizing tumors. In the case of these granular cell tumors I put the word "malignant" in front of granular cell myoblastoma to indicate its special potentials. This applies especially to the organoid granular cell myoblastomas, which are generally recognized as very malignant tumors.

CASE 7

Diagnosis.—Reticulum cell sarcoma (?) of forearm.

Contributor.—Dr. A. O. Severance, Baptist Memorial Hospital, San Antonio.

History.—A 23 year old man of Mexican descent injured his right forearm in November, 1954, and the area became swollen. This was thought to be a hematoma and was treated conservatively for about one month. Three days following a biopsy of the lesion the arm was amputated in the upper third. Gross examination revealed a yellowish-red, hard, lobulated tumor, 10 by 3 by 3 cm. This was separating and invading the flexor group of muscles in the forearm. The joints were not involved but the tumor was present about the periosteum of both forearm bones. A roentgenogram of the arm revealed no bone involvement.

Follow-Up.—Three weeks later a metastatic nodule was removed from the posterior cervical region. Postoperatively, roentgenograms of the chest were negative for metastasis on four occasions, the last film being made in April, 1955. The patient had left pleural effusion and died in May, 1955. No autopsy was performed.

Submitted Diagnoses.—Synovial sarcoma (malignant synovioma), 17; reticulum cell sarcoma, 8; rhabdomyosarcoma, 6; neuroblastoma, 4; neurofibrosarcoma, 2; sarcoma, unclassified, 2; Ewing's sarcoma, 1; "also ran," 3.

Dr. Stout: There comes synovial sarcoma, a favorite diagnosis, particularly when one doesn't know what else to call it. Then we have reticulum cell sarcoma, rhabdomyosarcoma, neuroblastoma, neurofibrosarcoma, sarcoma, Ewing's sarcoma, and three unnamed diagnoses. So the favorite diagnosis here is synovial sarcoma. We have next to show a roentgenogram of the forearm, and I'll ask Colonel Harrell again to interpret it.

Col. Henry C. Harrell, Fort Sam Houston: The reproduction here is quite accurate and there is no pathology that I can demonstrate either in the periosteum or the cortex of the bone. Bones and joints in both of these projections as nearly as I can tell are normal. The reproduction of the slide precludes study of the soft tissues.

Dr. Stout: So you wouldn't want to say whether

or not there was any tumor in the soft tissues, from this particular reproduction?

Colonel Harrell: No, sir. From the reproductions I can't see the soft tissues well enough, but I believe that the bones are normal.

Dr. Stout: This is a tumor composed of cords of moderately large rounded cells. The cytoplasm is not voluminous and seems irregularly vacuolated as if in degeneration. Mitoses are infrequent. With the Laidlaw silver reticulin stain there seem to be exceedingly few reticulin fibers intermingled with the cells. There is no evidence of secretional activity and no metaplastic activity. The tumor cells do not secrete mucin or hyaluronic acid and there is no evidence of epidermoid differentiation.

The interpretation of this tumor is not easy, and I do not think we can be absolutely certain of its nature since the patient has died and no autopsy was done. We have to decide whether the tumor is primary here in the soft tissue, whether it arose in bone and extended into soft tissues, or whether it was metastatic from a distance to the forearm. There is one tumor which could do this without making manifest its primary site. That is the seminoma of the testis. It would be unusual for a seminoma to behave in this fashion, but it is possible. The rounded cells of moderate size apparently without reticulin formation by the cells and growing in cords is not incompatible with seminoma. Further, a tumor of the testis may remain occult and still metastasize. If it is primary in the forearm it might have been a reticulum cell sarcoma of the bone marrow manifesting itself chiefly in the soft tissues; but the arm was amputated, so that the bones were probably investigated and no tumor found in either radius or ulna. No doubt some will want to consider the possibility of synovial sarcoma. The seminar tumor does not show the dual cellular composition of the synovial sarcoma so that I cannot agree to that diagnosis. This leaves us with reticulum cell sarcoma of the soft tissues for consideration. I would be willing to accept that as the most probable diagnosis were it not for the fact that there are practically no reticulin fibers among the tumor cells. I do not think this is enough to invalidate the diagnosis entirely, but I cannot make it with complete assurance.

I have not made any progress in determining the exact cellular nature of tumors of this sort. You will find a similar case in the proceedings of the seminar on tumors of the soft tissues of the American Society of Clinical Pathologists. I still think this is a distinct entity which can be primary in the soft tissues. I imagine some of the cases I have called by this name were metastatic and I was in error, but not all of them. After all, if everyone accepts the reticulum cell sarcoma of bone marrow, why should there not be one primary in the soft tissues, perhaps derived from histiocytes?

Perhaps it will be in order to elaborate the question of the necessity of demonstrating reticulin in a tumor before accepting it as a reticulum cell sarcoma. I don't believe that that is an absolutely necessary factor in the diagnosis. Let us take the question of Ewing tumor and reticulum cell sarcoma of bone. Ewing tumor does not make any reticulin fibers at all. Reticulum cell sarcoma, so-called, almost always does. Then what is a Ewing tumor? What cells does it come from? I got interested in that some years ago, and I tried writing around to various authorities, including Dr. Ewing, to find out what kind of a cell they thought the Ewing tumor cell was, and what it came from. There were two schools of thought at that time, and I suppose there still are. One was that the Ewing tumor comes from some undifferentiated cell of the bone marrow. Dr. Ewing thought that it was what he called a tumor of endothelial cells that form on the outer surface of capillaries. I tried hard to find out what that meant, and I never succeeded in getting Dr. Ewing to tell me exactly what he meant by perivascular endothelium; but at any rate he did not think that the Ewing tumor was derived from a cell similar to the cells of the reticuloendothelial system. On the other hand Oberling and several other people believed that the Ewing tumor cell was a derivative of a reticuloblast in an earlier phase of differentiation before it developed the ability to form reticulin fibers, so he believed that Ewing tumor and reticulum cell sarcoma of bone were simply different phases of derivatives of reticuloblasts. In the Ewing tumor the reproduction was in a very early phase and in the reticulum cell sarcoma the cell was further along in the differential series. If you accept Oberling's ideas, it is possible for what is called a reticulum cell sarcoma to fail to form any reticulin fibers; and I must say, in studying tumors that imitate the appearance, let us say, of reticulum cell sarcoma but which fail to form fibers, I can best explain them to myself by considering them all derivatives of reticuloblasts. So that I could accept this as a reticulum cell sarcoma of soft tissues. It is rather astonishing to me that so little has been written about reticulum cell sarcoma of soft tissues, and so much written about reticulum cell sarcoma of bone. After all, the soft tissues are really just as important as the bone, but nobody seems to take much interest in them.

Now in regard to the diagnosis of synovial sarcoma: I cannot persuade myself to make that diagnosis unless I see a tumor of dual nature, in which there are groups of cells resembling fibroblasts separating cells that resemble synovial cells either by forming glandlike spaces or slits, or by growing in solid cords, but without any reticulin fibers. In this tumor I can't see anything that I would be willing to pick out as a fibrosarcoma-like element. There is a

lot of reticulin, of course, but it all seemed to me to be simply part of the supportive framework of the tumor, so I am not willing to accept this tumor as a synovial sarcoma. I might say that I think it is a great pity that the term "synovioma" is also applied to tumors that could better be classified as villonodular synovitis, or giant cell tumors of tendon sheaths or joints, because to me they are completely different types of tumors. The true synovial sarcoma, strangely enough, develops in the tissues outside of the tendon sheath or joint, and if the tendon sheath, joint, or bursa is involved, it is by secondary invasive growth and not by primary involvement, in my experience; whereas of course the villonodular synovitis or giant cell tumor of the tendon sheath or joint usually develops right in the joint capsule or tendon sheath. It simply puts a lot of generally benign tumors in the same category as the very malignant synovial sarcomas, and I think that is too bad.

Dr. Leo Lowbeer, Tulsa, Okla.: Dr. Stout, since you mentioned in connection with reticulum cell sarcoma, Ewing's sarcoma, would you consider in this case the possibility of a metastatic sympathicoblastoma since in all cases of diagnosed Ewing sarcoma one should really suspect the possibility of a sympathicoblastoma primary in the adrenal gland?

Dr. Stout: Well, this patient is 23. The number of cases of metastatic sympathicoblastoma in adults, although a little more frequent than we used to believe, is still extraordinarily rare. I could see nothing in this which would guide me toward suggesting that diagnosis, because if you don't get some kind of differentiation like a genuine pseudorosette, I don't think that you could reasonably entertain the diagnosis. Moreover, I think these cells are too large to be probable candidates as sympathicoblasts.

Dr. Béla Halpert, Houston: Is it necessary, Dr. Stout, always to try to hitch any undifferentiated neoplasm to something known that is differentiated? Why couldn't we stop and say this is a mesenchymal cell tumor, undifferentiated? It didn't go far enough to show its character; consequently we can't classify it any further. I don't think it is good practice to call it a reticulum cell sarcoma when it doesn't make any reticulin.

Dr. Stout: I'll tell you why I try to do it. If you throw the gates open to the use of the term "undifferentiated sarcoma," you are going to have a hodgepodge of tumors put into that class, and it will never be possible to find out anything in regard to biological behavior from a hodge-podge of different kinds of tumors. The way I get around making a sure diagnosis on a questionable case is by making the diagnosis with a point of interrogation after it, because that indicates that I am not sure in my mind that the diagnosis is correct, but that I think that it might belong to the class named. I have done that now in a number of instances, and I am hoping to get all the cases of that nature together and analyze them to see what proof or disproof I can get toward this business of the reticulum cell sarcoma without reticulin formation. I don't know whether I will ever live long enough to find out, but I will try.

CASE 8

Diagnosis.—Reticulum cell sarcoma (?) of ileum with metastases to adrenals and left testis.

Contributor.—Dr. L. J. Manhoff, Jr., Robert B. Green Hospital, San Antonio.

History.—A 47 year old Latin-American man was admitted to the hospital one week before death with weight loss and fever of six weeks' duration. Numerous diagnostic studies were noncontributory. The patient suddenly developed an acute abdominal condition, and exploratory surgery revealed a large perforated ulcer of the ileum, which was resected. The gross specimen revealed an irregular perforation approximately 2 cm. in diameter which was in the center of a large, irregular ulcer measuring 4 by 5 cm. The patient died the following day of peritonitis. At autopsy large tumors were found replacing each adrenal gland, each measuring 10 cm. in diameter, encapsulated but adherent to surrounding structures. The masses were soft and on section were reddish brown with yellowish areas of necrosis. The left testicle was only slightly enlarged but was replaced entirely by similar tumor. There was no involvement of lymph nodes or any other organs. The slide includes a section of the ileum and adrenals. The testicle appeared similar histologically to the adrenal.

Submitted Diagnoses. — Lymphosarcoma (reticulum cell type), 15; lymphosarcoma (lymphocytic cell), 2; malignant lymphoma (unclassified), 2; seminoma, 8; embryonal carcinoma, testis, 6; sympathicoblastoma, 3; Hodgkin's sarcoma, 1; miscellaneous malignant lesions, 6.

Dr. Stout: The diagnoses lie largely between lymphosarcoma and a testicular tumor, as was to be expected.

Sections of the ileum show that the entire thickness of the wall is infiltrated by tumor cells which vary considerably in kind. Some are plasma cells, and multinuclear forms are present. Others which show mitoses are larger and a good deal more like reticulum tumor cells. A Masson trichrome stain shows many fine reticulin fibers scattered among the tumor cells. A mucicarmine stain shows no intracellular mucin in tumor cells. The gut wall is largely destroyed. The greatest concentration of tumor cells is toward the lumen, where there is superficial erosion of the tumor. The second slide, I presume, was taken at autopsy from one of the adrenals. Now the tumor no longer shows plasma cells and the growth is composed largely of rather small reticulum-like cells. Trichrome and Laidlaw silver reticulin stains show a framework of fibers which forms a meshwork with the tumor cells set among the reticulin fibers of the mesh.

When I first glanced at this case and learned that at autopsy tumor involvement of the left testis had been found, I assumed that the growth was a seminoma with unusual metastases. But after studying the case more carefully I have come to the conclusion that this was not so, but that the primary tumor was a malignant lymphoma of the ileum. There are sound reasons for this. In the first place the greatest ileal involvement is on the mucosal side, which favors a primary mucosal origin. In the second place the tumor cells are much more like reticulum tumor cells and the associated plasma and undifferentiated cells belong more to the lymphoma group than they do to any other group, including seminoma. Perhaps the most convincing supporting evidence is the reticulin meshwork in the interstices of which the tumor cells are found. Seminoma cells do not form any reticulin meshwork, but reticulum cell sarcoma can do so.

If I am correct in supposing this is a reticulum cell sarcoma primary in the ileum, its behavior has been very curious. The absence of any lymph node involvement at all is remarkable, and just as remarkable is the bilateral adrenal involvement and the focus in the testis. The cases which Marcuse and I studied had either regional node involvement or generalization or both. However, the longer you live the more peculiar and unexpected will seem the behavior and appearance of tumors.

CASE 9

Diagnosis.—Dysgerminoma of ovary.

Contributor.—Dr. A. O. Severance, Baptist Memorial Hospital, San Antonio.

History.—A 6 year old girl had a mass in the pelvis. At operation on November 2, 1953, the surgeon believed that the ovary was the primary site of the tumor because of its size. There was no involvement of the uterus, peritoneum, or intestine, but clinically the retroperitoneal nodes at the root of the mesentery and along the aorta were enlarged and thought to be involved. The gross specimen consisted of a tube and ovary that weighed 295 Gm. A fallopian tube 2 cm. in diameter was identified on one surface of the ovary. The mass measured 10 by 8 by 8 cm. and was smooth but lobulated externally. A light yellowish-gray, firm, moist tissue bulged from the cut surface. The patient had deep roentgen-ray therapy and died at home on January 13, 1954. No autopsy was performed.

Submitted Diagnoses.—Dysgerminoma, 21; lymphosarcoma, 7; round cell sarcoma, 3; granulosa cell tumor, 2; granulosa cell carcinoma, 5; malignant serous cystoma, 1; miscellaneous carcinoma, 3; lymphoma with possible dysgerminoma, 1.

Dr. Stout: The majority of you thought it was a dysgerminoma, some voting for lymphosarcoma. Oh, oh! Round cell sarcoma! I wish I could get hold of those people and ask them what they meant by round cell sarcoma—whether that is simply an escape diagnosis so they don't have to make a guess as to what it is, or what. Granulosa cell carcinoma and a malignant serous cystoma—well, that connotes that the growth was partly cystic, or formed cystic structures. I certainly didn't see any of those. Lymphoma with possible dysgerminoma—that also puzzles me. I don't know whether he means that his first guess is lymphoma and his second guess is dysgerminoma, or whether this is a lymphoma and a dysgerminoma. However, certainly dysgerminoma is the tumor diagnosis of the majority.

My first set of slides comes from the ovary. This is largely replaced by a tumor which is very cellular and is composed of rounded cells about twice the size of small lymphocytes, which grow in short slender cords. Reticulin fibers separate the tumor cords, but there are no reticulin fibers between the actual tumor cells. Mitoses are present and tumor cells are found in a large vein. There is no evidence of glandular or epidermoid differentiation and no evidence of cellular secretion. A second section shows the uterine tube entirely enveloped and engulfed by tumor cells. The lumen is maintained, but all of the villous mucosal projections are replaced by tumor cells. Here the tumor has a somewhat different aspect than in the ovarian section. The cells are in solid masses instead of cords, and scattered at intervals among them are solitary normal appearing reticulum cells.

If one had only to look at the section made from the ovary, I would be willing to accept this tumor as a dysgerminoma. It had developed in the ovary of a child; it is made up of cords of what might be considered as undifferentiated male sex cells comparable to the seminoma of the testis and incapable of secreting any hormones; and it has behaved like a malignant tumor. But when we inspect the section of tube invaded by tumor, it is different. It no longer grows in cords but in solid sheets; the cells appear smaller; and there are scattered through it a normal reticulum as if this were a lymphosarcoma. In this case we have more evidence in favor of a dysgerminoma, but it is disturbing to have it imitate a lymphosarcoma. One wonders after seeing cases 8 and 9 how it is ever going to be possible to be entirely confident about the diagnosis.

The fact that this patient was less than 10 years of age is disturbing, for one can find in the literature articles which deny that dysgerminoma occurs in the first decade of life. However, this apparently is not true, for Mueller and his associates in an analysis of 427 cases found that 28 girls or 6.89 per cent were less than 11 years of age. The youngest girl was 2 years, and 6 girls were 5 years of age or younger. It is, however, extraordinary to find one as young as this.

I presume we are justified in considering this a case of dysgerminoma, but I am not at all happy about the histological picture.

Dr. Béla Halpert, Houston: You are using the two

terms "seminoma" and "dysgerminoma" interchangeably, or did I misunderstand?

Dr. Stout: The application of the term "dysgerminoma," as I understand it, is made to tumors of the ovary that are composed of immature male-type sex cells that are comparable to seminoma cells in the testis. There are people like Dr. G. Teilum and others who consider the gonads as a whole group together and discuss all the tumors that can occur in the gonads in a single group. Do you think that they are different tumors?

Dr. Halpert: Yes, I do. A dysgerminoma similar to the one arising in the ovary may occur in the testicle. A seminoma has a definite morphologic pattern with streaks of lymphocytes within the stroma, and extensive areas of hemorrhage. None of these characteristics can be seen here.

Dr. Stout: Well, you would call this a dysgerminoma.

Dr. Halpert: Yes.

Dr. F. P. Bornstein, El Paso: I would like to comment on the nomenclature for a moment. When Robert Meyer* first described the tumor he called it "disgerminoma" and spelled it with an "i" apparently to emphasize the dual nature of this tumor. I don't know where the "y" got into this tumor in the first place.

Dr. Stout: Well, that just shows you how terminology gets garbled. I never read Robert Meyer's original article. I didn't know that at all. I'm glad you called our attention to it.

CASE 10

Diagnosis.—Arrhenoblastoma of ovary.

Contributor.—Dr. H. J. Schattenberg Clinical Laboratory, San Antonio.

History.—A 16 year old patient was a well developed young girl who presented marked hirsutism with a deep male voice. A fairly prominent beard was present. There was complete atrophy of the breasts. The clitoris was enlarged to almost the proportions of a small penis. The gross specimen consisted of a tumor measuring 14 by 22 cm. which was removed from the left ovary. On sectioning, this structure was seen to be cystic. The cysts were multilocular and filled with a straw colored fluid. Crowded to one side of the cyst ovarian structure was noted. Within this ovarian structure a lighter appearing mass was seen which could be identified as tumor tissue. This measured 8 by 10 mm.

Submitted Diagnoses.—Arrhenoblastoma, 38; granulosa cell tumor, 1; androblastoma tubulare lipoides, 1; Pick's tumor, 1; adenoma tubulare testiculare ovarii, 1; gynandroblastoma, 1.

*Meyer, R.: *Pathology of Some Special Ovarian Tumors and Their Relation to Sex Characteristics, Am. J. Obst. & Gynec. 22:697-713 (Nov.) 1931.*

Dr. Stout: One would guess that the diagnosis would be arrhenoblastoma, from the clinical features, and I am happy to see that one person thought it was androblastoma tubulare lipoides. This is simply a nonmalignant form of arrhenoblastoma. This (adenoma tubulare testiculare ovarii) is a tumor I don't know—certainly a nice name. A tubular adenoma, testicular type, of the ovary, I suppose that means. Then we have the gynandroblastoma; but certainly arrhenoblastoma is the popular diagnosis.

This tumor has a striking histological aspect. It is composed largely of cuboidal to columnar cells arranged in rather slender twisted cords. When the cells are columnar, they have the long axis of the cell at right angles to the long axis of the cord. In a few places only do the columnar cells have vacuoles which may have been filled with lipids. In one place the cell cords tend to lose their identity in a mass of spindle-shaped cells set close together as in a theca cell tumor. No interstitial cells are recognized. The cell cords are set in a fairly dense fibrous stroma.

Obviously this tumor was a masculinizing one. If one follows Teilum's classification, the ovarian tumors which contain male sex cells are called androblastomas. There is one which is composed of Sertoli cells and is called by Teilum androblastoma folliculare lipoides and by others folliculome lipidique. This secretes estrogen. It looks something like this tumor histologically, but all of the cylindrical cells are filled with lipids. Those tumors which secrete androgen and are masculinizing include arrhenoblastoma and the tumor which is variously called ovarian Leidig cell tumor, ovarian adrenal tumor, luteoma, and masculinovoblastoma. Here we get into all kinds of trouble, for we find that not only can Leidig cells sometimes contain lipids and grow in cords as here but they also can appear in more solid masses associated with spindle-shaped cells which, says Teilum, is more characteristic of the so-called adrenal rest tumor or luteoma of the ovary. A focus of this sort is present in this tumor. Not being intimately acquainted with the various stages of male gonadogenesis, I feel rather lost among the Leidig and Sertoli cells when they appear in the embryonal state. Adult interstitial cells I can recognize. Unfortunately they appear very seldom in the arrhenoblastomas. The androblastoma tubulare lipoides or Sertoli cell tumor of the ovary I can recognize when it is in a pure form. As far as I can tell in this tumor under discussion there may be both Sertoli and Leidig cells. Obviously the embryonal Leidig cells must predominate, for the evidence of masculinization is clear. Langley admits that there is a form of arrhenoblastoma in which are found both Leidig and Sertoli cells. I will therefore call this tumor an arrhenoblastoma of mixed type of the ovary.

I always have a terrible time with ovarian tumors of this sort, trying to sort out the various kinds of

cells of which they are composed. I am sure I haven't seen enough of them to have any clear conception of their distinguishing features. It seems to me that every one I come across has some new quirk in it that I never saw before, but I am willing to believe that this one is a tumor which can properly be called an arrhenoblastoma, as the majority of you have done. I have been interested in the reaction of people to Teilum's papers. I thought they were very helpful in sorting out these tumors. Other people have told me they thought they just made the confusion worse. I don't see how the confusion could have been made worse.

Dr. Béla Halpert, Houston: It seems to me that a Leidig cell tumor with active secretion has to have some evidence of secretion in the cytoplasm. In the cytoplasm of normal Leidig cells one sees crystalline, golden yellow, granular substance. A Leidig cell tumor, if it is to be identified by its product, should also have crystals in the cytoplasm of some of its cells. Sertoli cells are inside the seminiferous tubules, and are the supporting cells of the spermia, or are their precursors. I don't know whether their cytoplasm has any particular pattern. The nuclei are elongated, and there is hardly any cytoplasm there. A tumor composed of Sertoli cells would have the appearance of Sertoli cells in an atrophied testis with no spermatogonia present. The Sertoli cells are columnar cells with light cytoplasm and basally placed nuclei. The cells in this tumor resemble such Sertoli cells in some areas, being columnar, and having a nonstaining cytoplasm.

Dr. Stout: The only trouble is that arrhenoblastoma is an easy word to say when one is dealing with a masculinizing tumor of the ovary, but the histology of the different ovarian tumors that masculinize is so vastly varied and different that it is only a vague generic term in my experience.

CASE 11

Diagnosis.—Virilizing adrenal cortical adenoma.

Contributor.—Dr. F. C. Coleman, Santa Rosa Hospital, San Antonio.

History.—A 6½ year old white girl had been noted at the age of 2 to have an enlarged clitoris. It continued to grow, pubic hair began to develop, and the child had a masculine distribution of musculature. Physical examination showed wide shoulders and narrow hips, muscular thighs, axillary and pubic hair, and masculine voice. The laboratory found an excretion of 1,500 mg. of 17-ketosteroids in 24 hours. The patient was operated upon.

Submitted Diagnoses.—Adrenal cortical adenoma, 19; adrenal cortical carcinoma, 12; adrenal adenoma or carcinoma (take your choice), 4; ovarian hilar cell carcinoma, 2; adrenal rest tumor, 1; virilizing lipoid tumor, ovary, 2; arrhenoblastoma, ovary, 1; masculinovoblastoma (adrenal adenoma), 1; adrenal cortical hyperplasia, 1.

Dr. Stout: The first two are pretty confident that it was a cortical adrenal tumor, although there is a division of opinion as to whether benign or malignant. Then there is a halting opinion that it might be an adrenal tumor; then we come along to the idea of an ovarian tumor. I suspect these people didn't realize that the tumor that was removed was really an adrenal tumor. They weren't sure whether they were dealing with an ovarian or an adrenal tumor. That applies to all those who suspected the ovary instead of the adrenal. Well, I think this person will change his mind about the word hyperplasia when he sees the picture of the tumor. If it is hyperplasia, it is the biggest and noblest hyperplasia I ever saw.

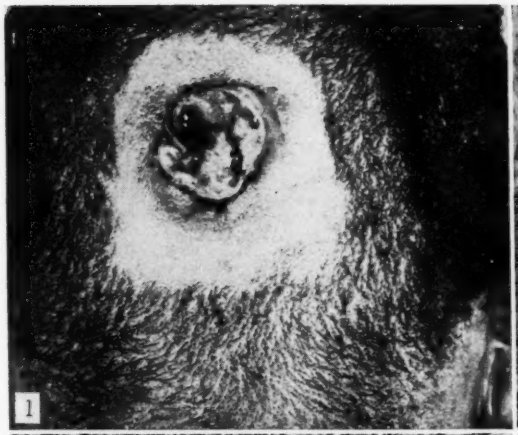
This tumor obviously reproduces accurately some of the layers of the adrenal cortex. A few giant cells with multiple nuclei are present. Most of the cells are well preserved, with granular cytoplasms. A few have vacuolated cytoplasms, and an occasional degeneration form is found. Secretory granules in some cells such as those described by Broster and Vines, and by Weber and Menten as evidence of virilization activity seem to be present in some cells. No mitoses are seen.

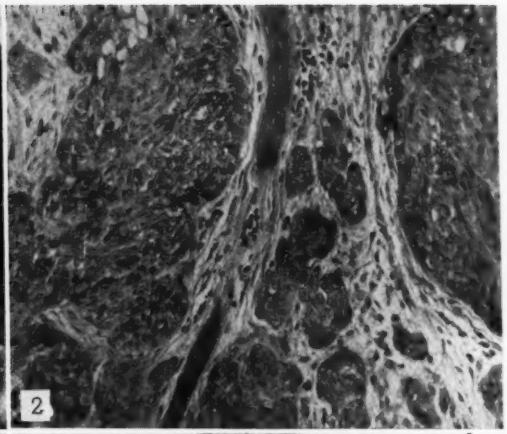
I have learned that this is an adrenal tumor. Certainly it has the characteristics of an adrenal adenoma so that I am not surprised. The question that must be asked is whether or not the secretory granules of Broster and Vines can be regarded as proof of virilization activity. I do not have any personal information on this subject, but if one is to judge from Karsner's statements, these cells can be found in normal adrenals and adenomas without any feminizing or virilizing activity. It seems that they are without diagnostic merit. Apparently it is not possible to tell by histological criteria alone whether or not an adrenal cortical tumor is producing sex hormones. There can be no doubt in this case that this adrenal adenoma was producing large quantities of 17-ketosteroids.

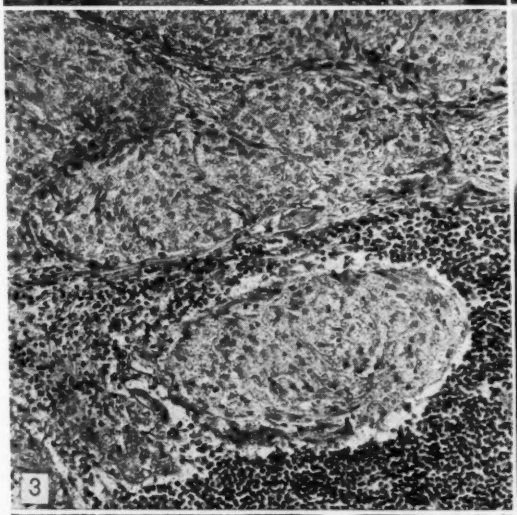
We want to know whether this is a benign or a malignant tumor—whether it should be called an adenoma or carcinoma. I do not know of any sure way of telling that. These cells are pretty well differentiated, and I think it is the general consensus of those who have written about these adrenal cortical tumors that unless you have marked evidence of active invasive growth, or malignant cell appearance, one cannot tell a benign from a malignant adrenal cortical tumor. We have no evidence in this case that the tumor is malignant, so I would favor the diagnosis of a virilizing adrenal cortical adenoma, realizing that I could be mistaken on the adenoma part.

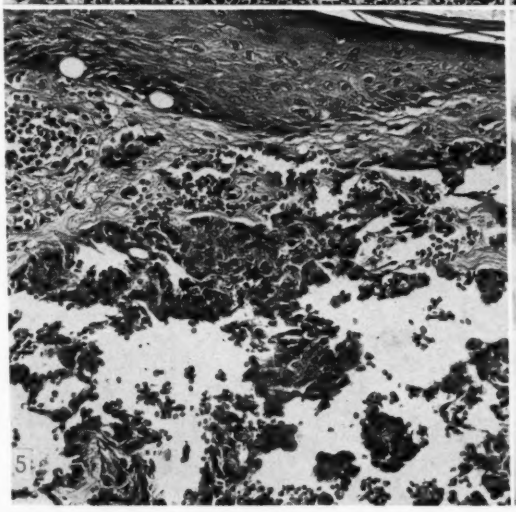
Does anybody have any thoughts on that?

Dr. Béla Halpert, Houston: I agree with Dr. Stout on the nomenclature of this tumor. I feel a little bit safer in regarding it as a benign growth because first of all it has an excessive secretory product. Second, it isn't invasive, but rather well encapsulated, and the









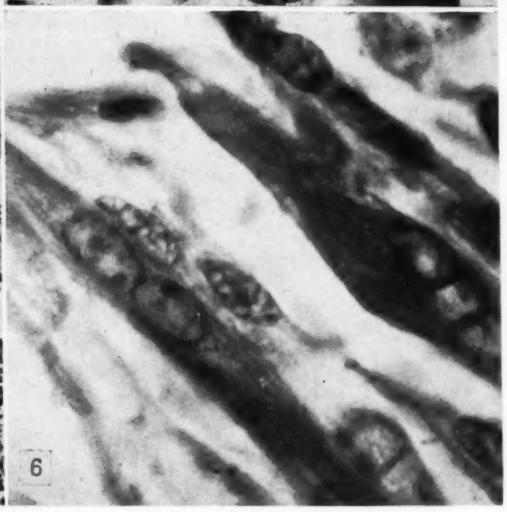


Fig. 1. Case 1. Carcinoma of scalp. Photograph before primary treatment.

Fig. 2. Case 1. Appearance of tumor invading corium. The central tube is a sweat gland duct.

Fig. 3. Case 1. Metastasis in cervical lymph node.

Fig. 4. Case 2. Appearance of tumor before resection.

Fig. 5. Case 2. Photomicrograph of tumor in skin. Anastomosing capillary vessels with intraluminal proliferation of tumor cells.

Fig. 6. Case 3. High power photomicrograph to show strap shaped rhabdomyoblasts with longitudinal intracellular myofibrils.

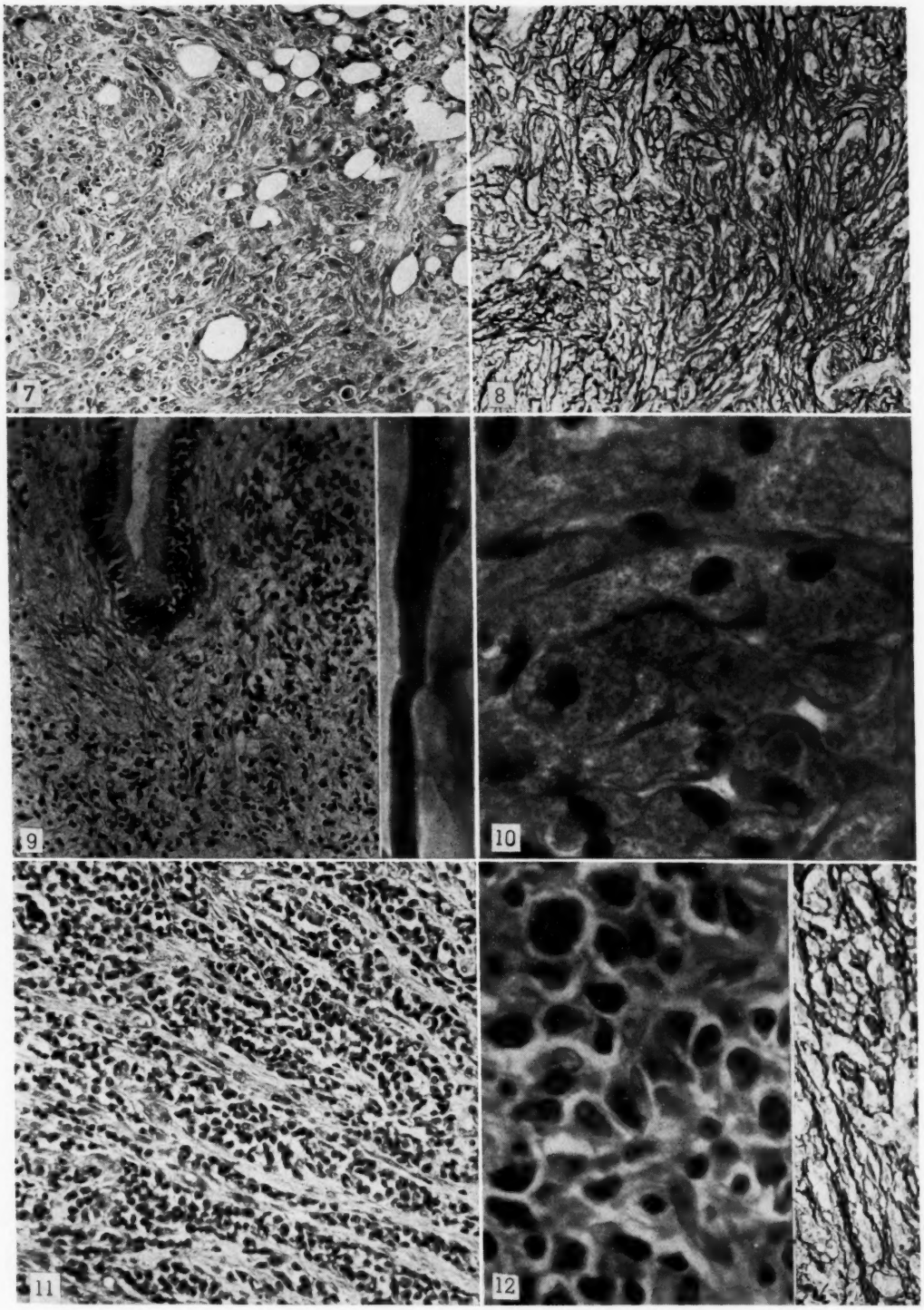


Fig. 7. Case 4. Photomicrograph of tumor invading adipose tissue of the mammary gland.

Fig. 8. Case 4. Laidlaw silver reticulin stain showing fibers surrounding almost every cell. From a more vascular area of the tumor.

Fig. 9. Case 5. Left. Photomicrograph from the myxoid portion of the tumor in the mucosa.

Right. High magnification of a strap cell showing cross striations.

Fig. 10. Case 6. Highly magnified tumor cells showing granules.

Fig. 11. Case 7. Invasion of fibrous tissues by strands of tumor cells.

Fig. 12. Case 8. Left. High magnification showing tumor cells. Right. Silver reticulin stain showing reticulin meshwork.

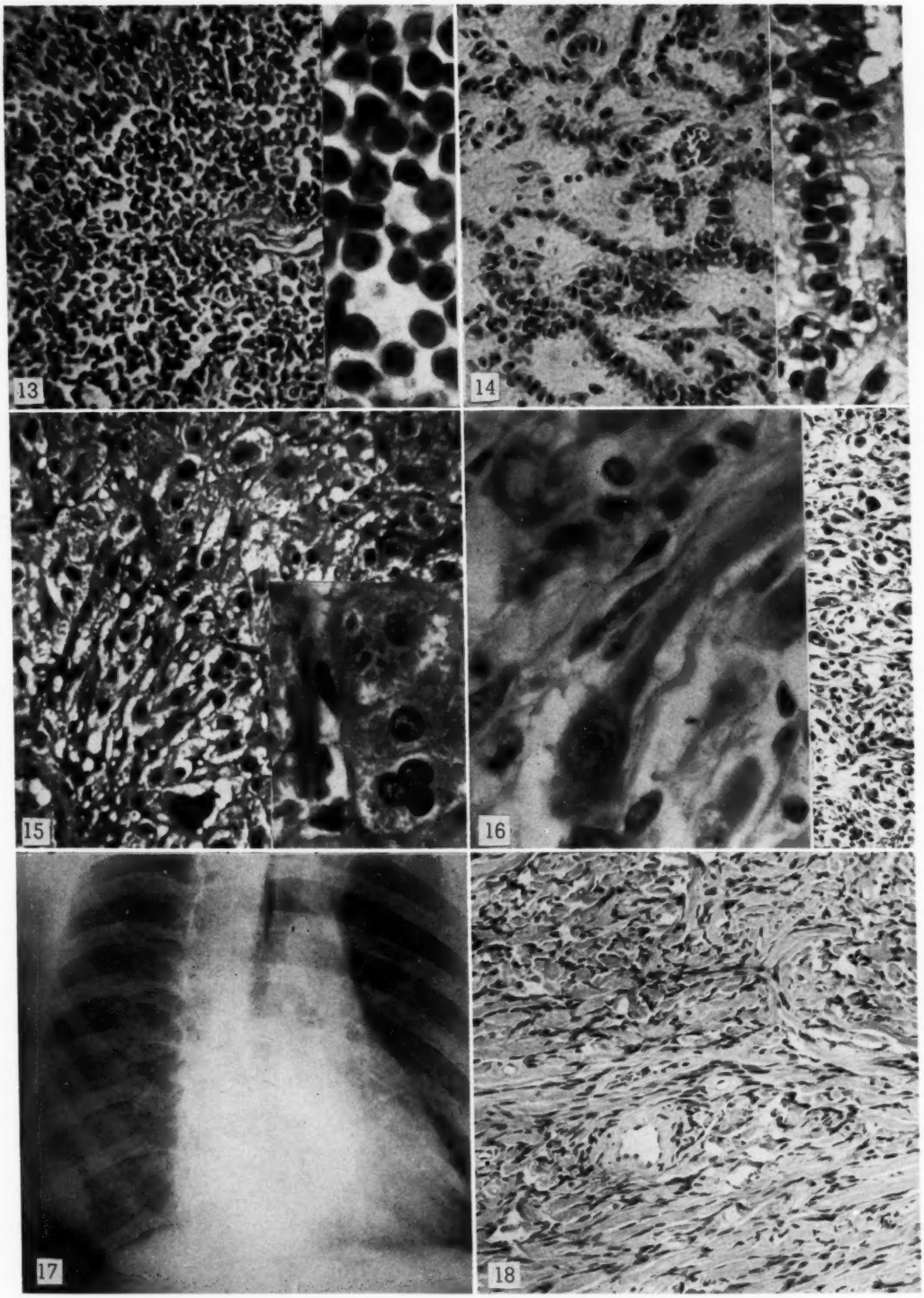


Fig. 13. Case 9. Left. grouping of the tumor cells.
Right. High magnification of individual cells.

Fig. 14. Case 10. Left. Cords of embryonal sex cells which do not have lipid containing granules (Leidig cells?).
Right. Cords of cells with cytoplasmic vacuoles (Sertoli cells?).

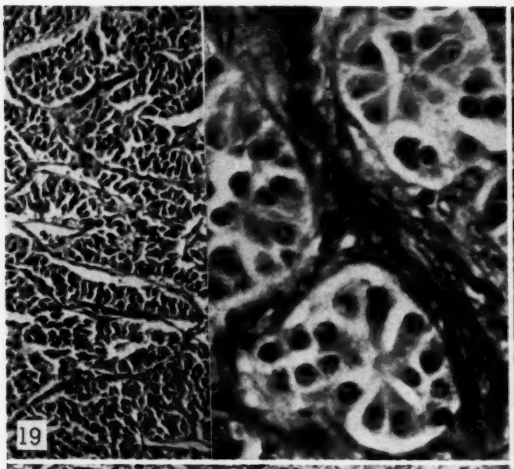
Fig. 15. Case 11. Tumor cells resembling caricatures of the adrenal cortical cells. The inset shows the granular cytoplasm of these cells and their close relationship with a capillary.

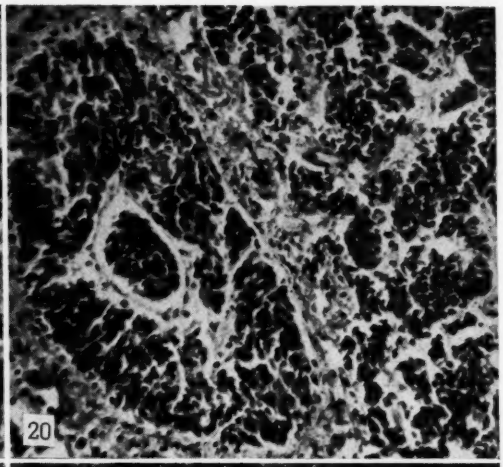
Fig. 16. Case 12. Left. Highly magnified tumor cells. The rhabdomyoblast in the center is racket shaped, and the head containing the nucleus has large peripherally placed vacuoles on one side of it. These probably contained glycogen.

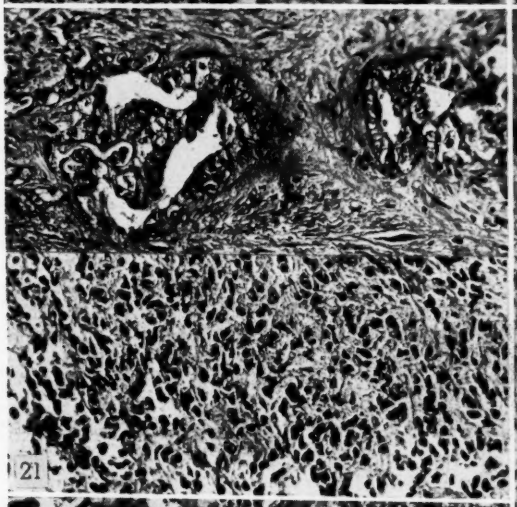
Right. The admixture of giant and elongated cells composing the tumor.

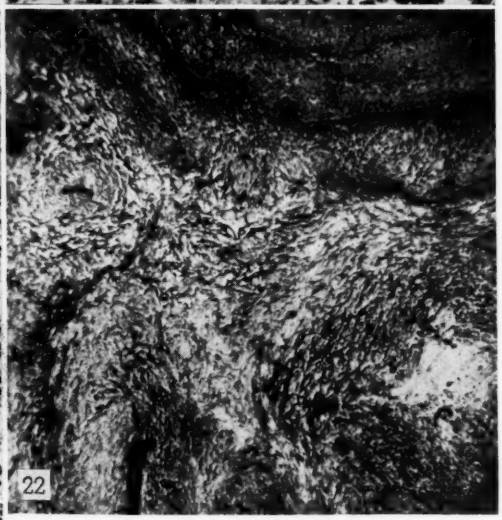
Fig. 17. Case 13. Roentgenogram of tumor which was pedunculated and attached to the posterolateral surface of the lower lobe of the right lung.

Fig. 18. Case 13. Photomicrograph of the fibrous tumor showing variability of cells and fibers.









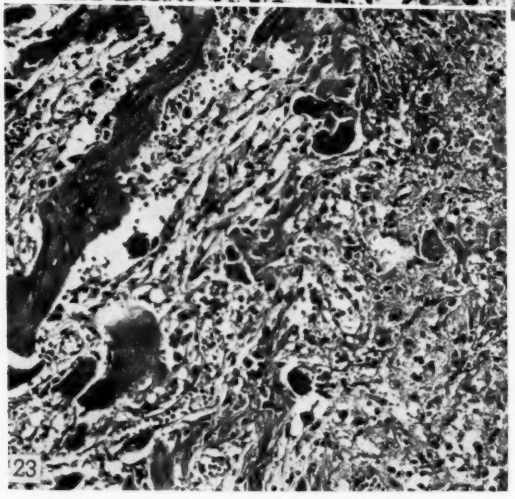


Fig. 19. Case 14. Left. The twisting cords of tumor cells separated sharply by fibrous bands.

Right. Two groups of tumor cells forming pseudorosettes.

Fig. 20. Case 15. Arrangement of undifferentiated tumor cells which form vaguely defined cords.

Fig. 21. Case 16. Differentiated glandular carcinoma above and sarcoma-like tumor cells below.

Fig. 22. Case 17. Peculiar grouping of cells and fibers about blood vessels.

Fig. 23. Case 18. Left. Partly destroyed bone trabeculae.

Right. Granulomatous tissue with multinucleate giant cells.

cells aren't distorted in any way. They are grouped just as nicely as one sees them in small cortical adenomas. I just wonder whether our intent on trying to differentiate malignant, that is cancerous growths, and so-called benign growths is not over exaggerated. Some years ago when I was talking with Dr. Hektoen he said that there are no satisfactory terms expressing the behavior of neoplasms. He used the term "cancerous" for "malignant." Perhaps we should emphasize certain qualities of the neoplasms that would indicate their behavior. A benign growth expands like a toy balloon; the malignant growth infiltrates, invades, and metastasizes. We could say of a neoplasm that it is invading, or that it is invading and metastasizing.

Dr. Stout: This, of course, is a tumor of an endocrine organ, and if you take all the tumors of the endocrine organs, I think you will find that in many instances a tumor with a benign aspect may surprise you by metastasizing. Take the islet cell tumors of the pancreas; I don't know any way to tell when one of them is going to metastasize and when one isn't. The reverse can be true. Take the paragangliomas—the nonchromaffin paragangliomas, if you choose, or the paragangliomas or pheochromocytomas of the suprarenal medulla; they can invade and do invade blood vessels freely, yet it is seldom that that results, in my experience, in fatal metastases. Maybe those cells that get into the veins do break off and go to the lungs or liver; but if they do, they must be destroyed there frequently, because of the infrequence with which actual metastases stop and grow. I haven't seen enough of the cortical tumors of the adrenal to have much opinion about them, but it is my understanding that it is possible to have an adrenal cortical tumor with all of the indications of being a benign growth which you have enumerated suddenly behave like a malignant tumor. So I always feel that one has to be a little cautious in being dogmatic about the expected behavior of one of these tumors of an endocrine organ.

Dr. Halpert: Once any of these have metastasized then there is no hesitation about them. As a matter of fact, I didn't want to take this up earlier in the morning, but when a basal cell tumor metastasizes, I would call it an undifferentiated squamous cell carcinoma.

Dr. Stout: An undifferentiated squamous cell carcinoma? Have you ever seen a basal cell tumor that metastasized? Come up to our laboratory and we will show you one that still looked like a benign basal cell tumor.

Dr. F. C. Coleman, San Antonio: I want to add a little follow-up. The six month follow-up on this patient showed a disappearance of the virilizing effects of the tumor except for the hoarseness. She did not show any evidence of recurrence, and her 24 hour excretion of 17-ketosteroids was 3.2 mg.

Dr. Stout: Well, it is good to get even a six months follow-up, although of course that isn't nearly enough to prove whether it is benign or malignant. I went so far as to make my diagnosis adenoma without any question mark on it, but in the discussion I sounded a note of warning that perhaps it might metastasize. If you sit on the fence on every tumor, it no longer becomes practical. You've got to take these things by the behavior of the majority of them showing the characteristics which the tumor under consideration shows. As Dr. Halpert pointed out, all the characteristics that we can observe in this tumor suggest that it is not a malignant tumor; therefore, I made the diagnosis of adenoma with the suggestion that it could be wrong, but I didn't put that suggestion in the diagnosis.

CASE 12

Diagnosis.—Rhabdomyosarcoma (malignant teratoma?) of testis.

Contributor.—Dr. E. Contreras Rodriguez, Mexico, D. F., Mexico.

History.—A 13 month old, well nourished boy was referred to the Children's Hospital because of a large testicular tumor of several months' duration. He was operated upon immediately, and a provisional diagnosis of seminoma was made. The gross surgical specimen was a large tumor involving the whole testicle and its coverings. Lymph nodes were not removed.

Follow-Up.—The patient was kept under observation until he was 5 years of age, at which time he was considered to be free of symptoms. He is still alive and enjoying excellent health at the age of 9.

Submitted Diagnoses.—Malignant teratoma (predominant rhabdomyosarcoma), 3; rhabdomyosarcoma, 12; benign teratoma, 5; malignant teratoma, 5; mesenchymoma, 5; myxoma (myxosarcoma), 4; embryonal carcinoma, 3; fibrosarcoma, 3; miscellaneous, 3.

Dr. Stout: Well, the majority called it a rhabdomyosarcoma. Colonel Farinacci, what does that mean with myxosarcoma in parentheses? Is that the way they expressed it?

Col. C. J. Farinacci, Fort Sam Houston: Well, a couple of them did, so I surmised that they meant myxoma, and therefore I lumped them together.

Dr. Stout: You possibly are aware that I don't know what a myxosarcoma is, so I never use the term. Thus the majority, but not everybody, thought it was a malignant tumor.

Two slides contain four sections. These show an embryonal type of immature testis with a large rete and a well developed epididymis. Associated with the testis is a tumor that is variable in its appearance; there are areas of rounded small cells, myxoid areas with stellate cells, one area with scattered large irregularly rounded or nodular cells with acidophil cyto-

plasm, and adjacent to them a few slender strap cells with acidophil cytoplasm, and finally fibrosarcoma-like areas. No epithelial elements are detected.

In this case the tumor so far as it can be observed in the sections furnished is a rhabdomyosarcoma and no differentiated or undifferentiated epithelial elements can be detected. If the tumor had been on a free surface like the bladder or perineum, it might have formed grapelike edematous areas like the sarcoma botryoides. Dixon and Moore do not recognize a primary rhabdomyosarcoma of the testis although they do recognize that such a tumor can develop in the lower part of the cord. They follow the general belief that if a rhabdomyosarcoma does form in the testis, it must be the unilateral development of one tissue in a malignant teratoma. This may be true, but I cannot see any reason for assuming that a rhabdomyosarcoma such as this may not have been a pure one-cell type of tumor. The sarcoma botryoides can develop in the prostate of a male; why not also in the gonad? The child at present is reported without evidence of metastasis, and well. If he should remain so, it seems to me an argument in favor of the sarcoma botryoides variety of rhabdomyosarcoma because that tumor type in the genitourinary organs seldom metastasizes while the teratoma of the testis metastasizes widely. In any event the tumor must be either pure rhabdomyosarcoma or the rhabdomyosarcomatous form of the malignant teratoma.

Dr. Béla Halpert, Houston: I called this a fibrosarcoma, and thought it originated in the epididymis. I was the first to describe one of these fibrosarcomas occurring in the epididymis,* and I thought this wasn't an exact image of it, but one which was close to it, particularly that interlacing pattern of the cells. However, your demonstration of cross striations shakes my confidence in that diagnosis. On the other hand, if one thinks of a myoblast as a mesodermal cell, it is possible for the cell which looks like a fibroblast to be a precursor of a cell which eventually develops striations. There is a stage of development in the wall of the gallbladder where no muscle cells are seen, but the fibroblasts seen are perhaps the precursors of the smooth muscle cells. Since the majority of the cells haven't acquired these striations I still would favor the diagnosis of fibrosarcoma of the epididymis. Those tubules are those of the epididymis and not of the testis.

Dr. Stout: I can't go along with you on the fibrosarcoma idea. If you open the door to fibrosarcomas producing cells of the aspect of rhabdomyoblasts, I think you are going to confound the diagnostician into an inability to predict properly what is going to happen to fibrosarcomas. I have spent a long time on that subject, too, and I believe that the explanation that you are suggesting can perfectly well be reversed. It seems to me that any of these special cell types can have areas of fibrosarcomatous aspect, and I believe that that attitude toward these growths has helped me very much in sorting out the liposarcomas, for example, with extensive fibrosarcoma-like areas, and calling them liposarcomas with the connotations that accompany liposarcomas. For example, the malignant fibrosarcoma which is purely fibrosarcomatous but made up of anaplastic cells with lots of mitoses is a very malignant tumor indeed, metastasizes easily, and generally kills the patient. On the other hand the metastatic rate and the degree of malignancy of the liposarcoma of the poorly differentiated variety is much less, and I think it has been of some use to separate them. As I say, it has helped me to do that, by suggesting that any of these mesenchymal tumors can revert to fibrosarcoma-like areas, that is, revert to just the production of connective tissue fibers, because that was probably the original function of most of the mesenchyme. You are taking the other aspect.

Dr. Halpert: I am going along with you, Dr. Stout, on this. I think that it is true that whenever even in small areas a characteristic is clearly discernible, that takes preference over the undifferentiated areas. In a carcinoma, for example, if we see a few acinar-tubular structures, we are justified in calling it a columnar cell carcinoma, or adenocarcinoma, or whatever name is used. If this is, and it seems that it is, a rhabdomyosarcoma, it is a tumor indeed very rare, and probably the first one seen. I must admit that I was uncomfortable about the diagnosis of fibrosarcoma because it just wasn't quite like the first one that I saw, or a second one which I have seen since. I am glad that your special stains demonstrated that it was not a fibrosarcoma, and I do not insist on calling it that.

*Halpert, B., and Thompson, W. V.: Fibrosarcoma of Epididymis, Arch. Path. 43:358-362 (April) 1947.

CASE 13

Diagnosis.—Benign solitary fibrous mesothelioma of the pleura.

Contributor.—Dr. Sylvia Johns, Nix Laboratories, San Antonio.

History.—A 50 year old white woman had a negative chest roentgenogram from a mobile unit survey in January, 1954. A similar survey film in 1955 showed a solitary peripheral lung tumor, and exploratory thoracotomy was done. When the chest was opened, the right lung was found to be free in the pleural space. There were no adhesions. A large, smooth, round, firm, very hard, pedunculated mass measuring approximately 1.5 inches in diameter was found arising from the posterolateral surface of the right lower lobe—actually from the junction between lateral and posterior basilar segments of the right lower lobe. A lobectomy of the right lower lobe was performed, and the postoperative course was uneventful. The gross specimen consisted of a globular, bosselated, encapsulated hard tumor

measuring 4 by 3.5 by 2 cm. It had a small tag of attached soft lung. On gross section, the nodule was solid and predominantly gray and fibrous with small reddish foci.

Submitted Diagnoses.—Benign solitary fibrous pleural mesothelioma, 25; pleural fibroma, 12; pleural fibrosarcoma, 1; neurilemmoma, 1; sclerosing hemangioendothelioma, 1; bronchogenic carcinoma, 1; leiomyoma of lung, 1; actinomycosis, 1.

Dr. Stout: The predominant suggestion is benign solitary fibrous pleural mesothelioma; pleural fibroma is practically the same thing; pleural fibrosarcoma is not the same thing. I disown that spelling [neurilemmoma] with violence!—and I am prepared to support that with lots of correspondence with Greek professors and other Greek scholars, as well as some of the older editors of dictionaries. Apparently the modern dictionary editors have not investigated the derivation of the word, but I will just draw a line through one "m" there.* Then we have a sclerosing hemangioendothelioma, bronchogenic carcinoma, leiomyoma, and actinomycosis! Now let us see the roentgenograms.

Col. Henry C. Harrell, Fort Sam Houston: It certainly would be nice for a radiologist—he could live a good life if he had that many pathological diagnoses from which to choose. This is the tumor in question. The outline is fairly discrete. The point that I think as a radiologist I should make first is the location, if it is possible to determine, and then some guesses at what it might be. The oblique film shows this lesion in a slightly different aspect and indicates that it is far posterior. That still does not tell us exactly where it is. Is it along the nerve distribution of the intercostal nerve; is it in the pleura; or is it in the lung? Oftentimes if it is of neurogenic origin, the little neural groove along the rib will be enlarged. That is not the case here. The old trick that you all know to differentiate whether it is in the pleura or lung, or whether it is in the parietal pleura or visceral pleura, is pneumothorax, which we do not have the benefit of in this case. Pneumothorax would separate it away from the parietal pleura if it were situated there. The other procedure is a laminographic study, which would also help us with the periosteum of the rib, and whether or not there is evidence of calcium in the lesion. The lesion lies very close to the parietal pleura, and in the differentiation most of the things considered when the diagnoses were given should be mentioned. It does have the characteristic shape of a pleural mesothelioma, which is sometimes multiple, but in this case, as nearly as we can determine, is single.

Dr. Stout: If this had been a neurofibroma and the rib were grooved, it would indicate that the nerve running along the rib was also involved. If, on the other hand, this were a neurilemoma, we would not see, I think, that grooving of the rib because the neurilemoma is an encapsulated tumor that enlarges the nerve only at the very site where it grows and doesn't extend within the nerve at any distance away from it.

Colonel Harrell: Well, I certainly would not disagree with that because I do not know enough about it. But if it is attached to the nerve at all there should be some periosteal reaction at the site. It might be very localized. Neurofibromas of course are multiple many times, and they usually occur nearer to the spine. This far peripheral on the nerve would be highly unlikely.

Dr. Stout: Yes, that has certainly been my experience. It would be nearer the spine, but it might be an intercostal nerve, as you say.

Now we have no gross picture of this, and I am not absolutely sure about exactly where it was attached to the pleura; but certainly it is a tumor which was attached to the pleura, probably the visceral pleura, and I believe that it almost certainly projected outward from the pleura into the pleural space. I'll show you why in a moment.

This is an exceedingly fibrous tumor, the surface of which is covered in part by somewhat swollen to flattened mesothelial cells. The attached lung tissue is not seen. The arrangement of the spindle, elongated, or almost rounded cells composing the tumor can best be described by the word patternless. They vary completely in numbers in any given part compared with an adjacent part, they run in various directions, and there are sudden changes from quite cellular to quite acellular areas. All of the cells have the appearance of well differentiated fibroblasts, and mitoses are not seen. Where the surface mesothelial cells are preserved and not rubbed off, the tumor cells impinge directly upon them.

The diagnosis here is easy for anyone who has seen these growths in the pleura. They look much alike both grossly and histologically, and the "patternless pattern" of the fibrous growths is not found elsewhere in the body except in connection with mesothelial surfaces. It is my belief that, in spite of the large number of collagen and reticulin fibers, these tumor cells are derivatives of mesothelial cells. The basis for this was the tissue culture observations of Margaret Murray upon the solitary malignant fibrous

*"*In regard to the spelling of neurilemoma with one m: Traditionally the word neurilemma has been spelled with two m's because the dictionaries have long been under the impression that the Greek word 'lemma,' a loosely applied sheath or bark, was the word from which it was derived. Actually, its proper derivation is from the Greek word 'eilema,' a closely applied sheath or covering. The French devised this name originally and their spelling 'nevrilème' with one m reflects this more accurate derivation. I was responsible for introducing the word 'neurilemoma,' spelling it with one m according to this derivation."*—Stout, A. P.: Neurilemoma of Sciatic Nerve, Cancer Seminar 1:8-9, 1950 (Proc. Seminar at Penrose Cancer Hospital, Colorado Springs, Colo., 1949).*

mesothelioma which grew only mesothelial cells when explanted in vitro.

The last time I looked up the records of our mesotheliomas of all kinds in the three cavities was in December, 1953, when we had 165 cases. These are recorded in table 2 from the paper I published in the *Archives d'Anatomie Pathologique* in 1954. This shows that one can divide these mesotheliomas in three different ways: whether they are solitary or diffuse, whether benign or malignant, and whether fibrous, tubular (including papillary), or a mixture of these two. In the pleura most of the tumors were either benign solitary and fibrous, projecting into the pleural cavity or into an interlobular space, or else they were malignant solitary and fibrous and were buried in the lung. The rare mixed varieties were of great importance to my conception that the fibrous

TABLE 2.—*165 Cases of Mesotheliomas Studied at the Columbia-Presbyterian Medical Center from July, 1919, to December, 1953.*

	Solitary			Diffuse		
	Fibrous	Tubular	Complex	Fibrous	Tubular	Complex
Pleura 88:						
Benign	40	4	2	2	0	1
Malignant..	25	1	0	8	4	1
Peritoneum 71:						
Benign	8	22*	0	0	0	0
Malignant..	7	7	1	2	23	1
Pericardium 6:						
Benign	0	0	0	0	1	0
Malignant..	0	0	0	2	3	0
Total 165:						
Benign	48	26	2	2	1	1
Malignant..	32	8	1	12	30	2

*Epididymis 11; uterus 5; tube 3; colon 1; pelvic peritoneum 2. (Modified from Stout, A. P.: Les Mésothéliomes de la plève, du péritoine et du péricarde, Sem. des Hôp. de Paris, Arch. d'Anat. Path. 30:A-115-A-119, 1954.)

tumors of these serous surfaces were really derived from mesothelial cells, for the inclusion of the more familiar tubular mesothelial structures in fibrous tumors seemed to lend support to the hypothesis that both of them could develop from mesothelium.

These tumors which project out into the pleural or peritoneal cavities are fibrous, they have the characteristic patternless pattern, and they are benign. I know of only one case which may have been related to a malignant tumor. This was in a man from whose visceral pleura a tumor of this sort was excised with a bit of lung attached. Seven years later a malignant solitary fibrous mesothelioma developed beneath and attached to the cicatrix in the lung and pleura from which the original tumor had been removed. I presume we must suppose the two tumors were related and that this type of removal has a certain hazard. It is so remote, however, that I think the morbidity connected with lobectomy might counterbalance the danger of recurrence in malignant form, making simple excision the operation of choice.

CASE 14

Diagnosis.—Bronchial adenoma (carcinoid type) of lung with metastasis to a hilar lymph node.

Contributor.—Dr. F. C. Coleman, Santa Rosa Hospital, San Antonio.

History.—A 26 year old woman had a routine roentgen examination of her chest which revealed an asymptomatic, round, opaque lesion located in the lower lobe of the right lung just above the right cardiophrenic angle. Physical examination revealed no other abnormalities. A lobectomy of the right lower lobe was performed.

Within the lung tissue, 1.5 cm. from the posterior end of the resected bronchus, was a well circumscribed, round tumor measuring 4 cm. in diameter. On section the tumor had a pale yellow-brown color. Distal to the tumor were several irregular cavities filled with necrotic gray-brown material. A small, oval lymph node, 1.2 cm. in diameter, which appeared to be involved by tumor was removed from the right hilar region.

Follow-Up.—The patient has been well, without any evidence of recurrence.

Submitted Diagnoses.—Bronchial adenoma (carcinoid type), 39; adenocarcinoma of lung, 2; metastatic carcinoma, 1; metastatic carcinoid, 1.

Dr. Stout: I had supposed the diagnoses would be nearly unanimous. Now let's have the roentgenograms.

Col. Henry C. Harrell, Fort Sam Houston: Without benefit of all those astute diagnoses from the chest men, I'd like to point out that it is not nearly that obvious with roentgen ray. Here we have a rounded lesion and there is no area of lung between the lesion and the heart, which would place it anterior in the chest. It is fairly solid in appearance, and there is no calcium that I can see in it. There is also no atelectasis that can be seen, with the possible exception of a slight amount in the middle lobe. The lateral film shows the lower boundary of the mass, and there is some atelectasis that goes posterior in the lower lobe instead of the middle lobe. From the radiologic point of view we just have to differentiate a solid mass, and from the age of the patient we could start out with an adenoma. If the patient were a little older, there is no way I know that one could tell for sure whether this is an adenoma or a bronchogenic carcinoma.

Dr. Stout: Is that atelectasis enough to make you feel fairly confident it is a bronchial growth?

Colonel Harrell: Yes, sir, I think I could be fairly certain of that.

Dr. Stout: Now we have a photograph which is a pretty picture of the tumor; but the cut has been made in such a way that I can't figure out where this is in relation to the hilus. It seems to be extending out into a bronchus, and probably largely obstructing it, because I think this is a little bit of

bronchial space here. You can see that it extends well out toward the pleura, and quite far in, but what its definite relationship with the bronchi and the hilus may be, I am not sure. We have to go along with the roentgen-ray picture that it probably is obstructing a bronchus. Now we will proceed to look at the microscopic appearance of this growth, which is striking.

This tumor has the characteristic appearance of the bronchial adenoma of carcinoid type. The cells usually are arranged in rather large rounded or ovate clusters with fibrovascular septums separating the clusters. The cells vary from rounded to elongated; the cytoplasm is filled with very fine granules. Occasionally the cells form rosettes. There is little variation from this picture, and mitoses must be rare, for I have not found any. The most striking variation from the usual histological picture is the presence of groups of tumor cells in a considerable number of endothelial-lined tubes. Some of these are probably capillaries, for they communicate with capillaries and occasional isolated red blood cells are found in them. Whether or not any of them are lymphatics I do not know. In places the tumor cell clusters have "shrunk away" from the surrounding fibrous tissues as so often occurs in carcinoid and islet cell tumors. A small amount of differentiated bone is found buried in one part of the tumor.

The picture is characteristic of the carcinoid variety of bronchial adenoma. The interesting feature is the fact that a metastatic deposit was found in one of the bronchial lymph nodes. The most extensive study on the incidence of metastasizing bronchial adenomas comes from the Mayo Clinic in a paper by McBurney, Kirklin, and Woolner. From April, 1937, through January, 1952, there were 111 bronchial adenomas verified histologically; of these 9 metastasized. Including their own and those in the literature, 87 cases with metastases were found. Forty-six were in the regional nodes alone. Of the 41 remaining cases metastases were found in the following organs in a decreasing order of frequency: liver, opposite lung, ipsilateral lung, pleura, esophagus and other mediastinal structures, and a scattering of organs including brain, kidney, and adrenal. These authors think that about 10 per cent of bronchial adenomas metastasize and that proportionately there are more frequent metastases from the cylindromatous adenomas, but of course these are rare forms of bronchial adenomas. They could usually detect some unusual changes in the adenomas that metastasized such as anaplasia or increased mitotic rate; where, however, metastasis went only to the regional nodes, they could not detect any differences from the ordinary adenoma in 2 cases. This present case is unusual because of the presence of tumor cells in capillaries and perhaps in lymphatics. It is of some interest that at the Presbyterian Hospital we have not had any cases of bronchial adenoma which metastasized, so far as I am aware.

Incidentally, I think it is a pity that the cylindromatous tumors should be classified with the carcinoid type of adenoma. For me the cylindroma is a carcinoma and should be called a cylindromatous carcinoma rather than adenoma. There are such tumors as simple mucous adenomas of the bronchial mucous glands. They are quite benign. They are extremely rare.

Let me ask one question. Would anybody who has seen a metastasizing bronchial adenoma hold up his hand?—exclusive, of course, of this case. Well, quite a number have—about 10.

CASE 15

Diagnosis.—Undifferentiated carcinoma (?) of anterior mediastinum.

Contributor.—Dr. E. Contreras Rodriguez, Mexico, D. F., Mexico.

History.—A 50 year old man developed rapidly progressive facial edema and dyspnea. A huge mediastinal tumor was shown by roentgen ray and proved to be radiosensitive. In spite of intensive treatment the patient died. At autopsy the main mass was in the anterior mediastinum involving the great vessels and the upper pericardium. It extended toward the trachea and bronchi without important distortion. There were metastases to the mediastinal and mesenteric lymph nodes and to the lungs and liver.

Submitted Diagnoses.—Bronchogenic carcinoma, anaplastic, 17; thymoma, 9; malignant thymoma, 10; spindle cell carcinoma, thyroid, 1; squamous cell carcinoma from a mediastinal teratoma, 1; mesenchymoma, 1; ¿Quien sabe?, 1; sympathicoblastoma (neuroblastoma), 2; reticulum cell sarcoma, 1.

Dr. Stout: Well, we have bronchial carcinoma, thymoma, and malignant thymoma, so we have 19 thymomas to 17 bronchogenic carcinomas, and then a scattering of other diagnoses. Now we have some roentgenograms.

Col. Henry C. Harrell, Fort Sam Houston: This probably is amusing to everybody but the patient, and believe me, when the tumor looks like this radiologically, it is not amusing to the patient. This is a big tumor and it appears to be infiltrating. It looks like it is growing out into the lung in all directions. I cannot make out the trachea very well, but I think it is a little displaced. The tumor appears to have arisen in the mediastinum some place. There is little atelectasis here. The minor fissure is not displaced as one would expect if a large portion of the lung were atelectatic. There is a moderate amount of emphysema below it. The diaphragm is lowered and straightened, which is compensatory for the amount of tumor that occupies the upper lung.

The anterior margin of this large tumor can be located more or less in the anterior portion of the

mediastinum. Most of the tumor lies laterally to the trachea, but then it comes anteriorly also. There is not much evidence of atelectasis. It is all infiltrative.

As far as the differential diagnosis is concerned from the roentgenologic point of view, most of these tumors demonstrating this appearance will be bronchogenic carcinomas. As far as the malignant thymoma, which was a popular diagnosis, is concerned, I certainly could not say from the radiologic appearance that it is not one; but in my experience the thymomas have been distributed in a little different manner. They can come out of the mediastinum in any direction, of course, but the ones I have seen, which have been few, have been further down in the mediastinum. Bronchogenic carcinomas also can occur anywhere, but I think that from a roentgenologic point of view I should say this is probably a malignant tumor arising near the mediastinum. Bronchogenic carcinoma would be my choice.

Dr. Stout: Have all of the thymomas that you have known to be such been anterior and just behind the sternum? Have you ever seen one that wasn't?

Colonel Harrell: No, sir, I do not believe I have seen one in the posterior mediastinum, and low, either. The few I have seen have been mostly in the anterior and lower portion of the mediastinum.

Dr. Stout: The following is the gross autopsy report:

In the thoracic cavity, the right pleural cavity presents adhesions, firm in the upper part; after these are broken, 250 cc. of serosanguinous fluid is found. In the left pleural cavity there are also firm adhesions and the same type of fluid in the quantity of about 500 cc. The pericardial sac is very thick and contains 500 cc. of straw colored fluid. In the posterosuperior mediastinum there are several masses of tumor-like aspect of a grayish-white color, soft, that extend toward the right lung and to the front, enclosing the great vessels and invading the atrial walls. In the abdominal cavity there is a small amount of straw colored fluid. There are a few adhesions in the splenic angle of the colon. After these are broken there is found a tumor-like nodule in the diaphragm. Some mesenteric lymph nodes are moderately enlarged.

In the study of the organs, in particular the gastrointestinal tract, there are not found any important alterations. In the lesser omentum and in the edges of the pancreas there are several enlarged lymph nodes which are firm. The pancreas weighs 110 Gm., and on multiple cut sections it presents a nodule with a tumor aspect in the parenchyma about the head of the pancreas. The liver weighs 1,960 Gm. Its surface is smooth and purple with yellowish discolorations. In the right lobe there are two neoplastic nodules, and there is one more in the Spiegel lobe. The right main bronchus is filled with a purulent material. Both lungs weigh 2,300 Gm. The left lung is seeded by tumor-like nodules, and on cut section the parenchyma appears congested. The right lung presents marked congestion in its middle and inferior lobes. The superior lobe is very large and presents costal indentations with marked thickening of the pleura. On its sagittal section are found gray hepatization of all of the superior lobe, and a tumor nodule, apparently extrapleural, in its inner margin. In the circulatory system the neoplastic invasion of both atriums and the involvement of the great vessels by the tumor have been mentioned. Excepting these, the heart is not remarkable in its cavities. The aorta presents plaques of atheromatous calcification. All the lymph nodes around the aorta and in the abdomen, as well as in the thorax, are enlarged and have a tumor-like aspect. The urinary bladder, kidneys, and endocrine glands are not remarkable. Both testes were said to be normal. The spleen is atrophic, weighing only 60 Gm., and on cut section large areas of fibrosis can be seen. In the central nervous system cut sections are not remarkable.

A large number of sections were furnished me, made from various parts of this tumor and its metastases. They all show a comparable picture, and there is no essential variation from one part to another. The tumor cells grow in well defined and generally rather slender cords. The individual cells are predominantly oat shaped, but some appear rounded. They are anaplastic and do not appear to secrete anything nor show any metaplastic glandular or epidermoid differentiation. In one or two places I thought I could detect the formation of pseudorosettes, but I could not be sure, and since there is no decided evidence of this I could not accept it as significant. Mitoses seem frequent and there is a more-or-less marked tendency for the cells to become necrotic.

A tumor in the anterior mediastinum may be a thymoma, a benign or malignant teratoma, a thyroid or parathyroid tumor, occasionally some of the uncommon forms of sarcoma or benign mesenchymal neoplasms, a malignant lymphoma, a nonchromaffin paraganglioma of an aortic body, a neuroblastoma, or an oat-cell carcinoma either metastatic from the lung or primary from foregut tissue in the mediastinum. There are probably some others which I have forgotten, but there are enough named to permit discussion.

I am willing to exclude thymoma because this is a single cell type and also because it has produced blood-borne metastases, which Castleman says do not occur in cases of thymoma. It bears no resemblance to tumors of the thyroid, parathyroid, or aortic bodies, so that I am willing to exclude these from consideration. All benign tumors can be excluded, including benign teratomas. I do not know of any malignant lymphomas which look like this tumor with its tendency to grow in cords; I will therefore exclude that group. This leaves a malignant tumor derived from one element of a teratoma; a neuroblastoma (sympathicoblastoma); and a metastasis from another site, possibly a primary oat-cell carcinoma of the lung.

Woolner, Jamplis, and Kirklin have described 3 cases of seminoma of the anterior mediastinum. In 2 of them the seminoma was the only finding; in the third case the seminoma was part of a teratoma. I cannot believe the tumor under discussion is a seminoma, for the cells composing it are quite different from the gonadal germinal cells. Can it be a neuroblastoma? This is distinctly a possibility. The neuroblast is not always rounded; it may be oat shaped as here. The cells as far as I can judge from the hema-

toxylin-eosin sections do not have any fibrils associated with them, and the marked tendency to necrosis is often seen in this tumor type. The age of the patient is against it, although it is not impossible for a middle aged adult to have a sympathicoblastoma. The anterior mediastinum is an unusual place, but again, not impossible. It could come from the neurogenous tissues of a teratoma and overgrow everything else. This I cannot exclude, but without some clinching evidence cannot accept. Finally we must consider an oat-cell carcinoma, either primary in the lung and metastatic in the mediastinum, or primary in the mediastinum growing from the out-pouching anlage of the foregut which eventually forms the bronchi and lungs. The histological features of this tumor could equally well apply to the oat-cell carcinoma as to the neuroblastoma. I spent some time looking for some differentiating features which might enable me to be certain this was an oat-cell carcinoma, but I failed. Therefore I cannot decide definitely between neuroblastoma and oat-cell carcinoma. We do not have an accurate description of the tumor in the lung so that I can form no opinion as to whether or not any of the areas of lung involvement might be considered primary. We have to presume that whoever did the autopsy did not think so at the time when it was done. Purely on the basis of probability I favor an oat-cell carcinoma derived from a teratoma or from a foregut anlage in the mediastinum, but it might be some one of the other explanations I have suggested, or something I have not thought of.

CASE 16

Diagnosis.—Collision tumor of gallbladder. Carcinoma differentiated of gallbladder. Malignant mesenchymoma (?) of gallbladder.

Contributor.—Dr. A. O. Severance, Baptist Memorial Hospital, San Antonio.

History.—A 67 year old white woman had a one week history of pain in the right upper abdominal quadrant. She was obese and moderately jaundiced. A mass was thought to be palpable in the right upper quadrant, and at surgery a tremendous tumor of the gallbladder involving the liver and splenic flexure of the colon was found. This was inoperable because of deep infiltration into the liver. The gross specimen consisted of a gallbladder 15 cm. long and ranging from 3 to 8 cm. in diameter. It was attached to a 3.5 cm. piece of liver which was replaced by a glistening gray tumor. A single large stone was found in the lumen of the gallbladder. The mucosa of the gallbladder showed a friable papillary soft tumor growing from multiple sites. It was also extending through the wall, and in some areas it had a gross appearance of fat. The patient died at home five weeks after surgery, and no autopsy was performed.

Submitted Diagnoses.—Adenocarcinoma of gallbladder, 24; carcinosarcoma, 11; combined adenocarcinoma and sarcoma, 3; squamous cell carcinoma, 1; adenocanthoma, 1; rhabdomyosarcoma, 1; liposarcoma, 1; small cell sarcoma, 1.

Dr. Stout: We'll consider the appearance of the gallbladder. It is laid open and the tumor partly projects out into the lumen of the gallbladder. Some of it is nodular and perhaps a little ulcerated, or eroded, on the surface. A cut of the gallbladder wall shows a thick muscularis. I don't know whether there is tumor invading outside of the gallbladder or not, but in any event it can be seen that there is a large area of tumor in the gallbladder.

This section shows a portion af the gallbladder wall. In one area the mucosa is replaced by a fungating tumor which is made up of masses and strands of completely undifferentiated anaplastic cancer cells. They do not form glands, and the mucicarmine staih shows no evidence of mucin secretion. This part of the tumor is limited to the mucosa and the superficial part of the thickened edematous submucosa. In the muscle coat and the deepest part of the thick submucosa are strands of well differentiated cancer cells which form bizarre glands, and their cells show evidence of mucin formation with the mucicarmine stain. There is no evidence in this section that the two carcinoma variants are in contact.

For some reason which I do not understand the gallbladder is an organ which develops carcinomas that are apt to differ in histological characteristics in different focal areas. There are not infrequently glandular and squamous areas in different parts of the same gallbladder or, as in this case, separate differentiated and completely undifferentiated foci in the same organ.

Tragerman reviewed 173 cases of gallbladder carcinoma from the Los Angeles General Hospital. He classified all the tumors as adenocarcinomas. Among these there were 22 classified as mixed types of adenocarcinoma and 6 more in which there were both squamous and adenocarcinomas. That is, about 16 per cent of the cases he studied had more than one type of carcinoma.

This case 16 had a large stone in the gallbladder. The statement has been affirmed and denied that all carcinomas of the gallbladder are associated with stone. Tragerman reports that he found stones in 138 of the 173 cases, or 79.8 per cent. That conforms with the findings in our cases. The relationship of gallstones to the etiology of carcinoma is discussed by Tragerman. No conclusion about this could be reached because of conflicting data. The results of attempts at treatment are generally disappointing, and few cures are reported. Tragerman has only 1 patient alive and well 12 years after operation.

The discussion and conclusion reached in this case was based upon the tissue submitted to the group for the seminar. By a devious grapevine it was brought to my attention that I had seen slides from this case two years before and had expressed a different opin-

ion from that which I reached from a study of the seminar slide. After investigation I found the slides sent me two years ago by Dr. Severance. I can assure you they looked very different from the seminar slides. What I have assumed to be an undifferentiated carcinoma looks much more like a sarcoma with myxoid areas containing stellate and spindle shaped cells, some of which have foamy cytoplasm containing lipids. There are other areas where the cells suggest rhabdomyoblasts but without any proof that they are. The disturbing thing about this tumor, however, is that it also contains areas like the one in the seminar slide which suggest carcinoma so that one feels confused and in doubt about the nature of the sarcoma-like portion. The differentiated carcinoma is obvious. The two tumors are in contact in one section, but they do not intermingle. We have, therefore, the choice of calling this gallbladder complex a collision tumor composed of two different varieties of carcinoma, one of which shows marked sarcomatous metaplasia, or a collision tumor made up of independent carcinoma and malignant mesenchymoma which are in contact but do not intermingle. In any event this cannot be a carcinosarcoma, for the two tumor forms are not intermingled so as to form a single tumor with both carcinomatous and sarcomatous elements. Probably the latter point of view is preferable since we know nothing about the capabilities of the gallbladder carcinoma cell to perform such miracles of metaplasia.

I'd like to ask Dr. Severance what he thinks about it, because this is his case.

Dr. A. O. Severance, San Antonio: I think that this is a carcinosarcoma, and I think it fits some of the criteria you have just elaborated upon. In the study of all areas and sections through this tumor, some areas are straight adenocarcinoma without any sarcoma elements. Some elements deeper in the wall are just plain sarcoma; and you can find areas where there are cells filled with vacuoles that stain with Scharlach R and that look like lipoblasts. Now you might say maybe that is degenerative change—the cells picking up fat from degeneration; that is a debatable point. Some areas look almost like the cells had cross striations, but I couldn't prove it. Other areas are very myxomatous, and you might worry about a myxoma. Other areas are just fibrosarcoma. Now in some areas of the tumor the sarcoma infiltrates, if you want to use that term, or intimately associates itself with the glandular part, with adenocarcinoma, and in these sections I thought each slide showed both types of tumor, because we looked at the beginning of the cutting of the block and at the end of the cutting of the block, and both types of tumor should be present in all slides.

Dr. Stout: That is, intermingled?

Dr. Severance: Yes. You got the best deal, though; you got the best blocks two years ago, and then when it came to cut for the seminar I had only what was left.

Dr. Stout: It couldn't have been the best block, because in my sections they weren't intermingled. They were in the same section, but they were not invading one another.

Dr. Severance: Well, they were very close together—right smack against each other.

Dr. Stout: Oh, yes, but that is the collision tumor —the kissing tumors.

Dr. Severance: May I be permitted to retain my original opinion?

Dr. Stout: O.K.

Dr. Béla Halpert, Houston: I agree with Dr. Stout that these are two different tumors which have met, that they are a carcinoma and a sarcoma. I have published a case of carcinosarcoma of the mammary gland where the stroma was sarcomatous and the parenchyma was the carcinoma. Such tumors exist in the mouse and in other animals too, so that the difference is just what you said. In carcinosarcomas they are intermingled; the sarcoma forms the stroma for the carcinoma.

Dr. Stout: Yes. Do you subscribe to the idea that the sarcomatous part is metaplasia of a carcinoma?

Dr. Halpert: I don't think so.

CASE 17

Diagnosis.—Hemangiopericytoma with fibromatosis (?) of thigh.

Contributor.—Col. Charles J. Farinacci, MC, Fourth Army Area Medical Laboratory, Brooke Army Medical Center, Fort Sam Houston.

History.—A 22 year old white woman had an asymptomatic mass in the right adductor canal, which was accidentally noticed at the time of her last pregnancy examination, two months prior to biopsy. Pregnancy was not interrupted. A wide surgical excision (hip disarticulation) was performed one week after biopsy.

The specimen consisted of the right thigh, which had been longitudinally and transversely sectioned. Between the vastus intermedius, the sartorius, and the adductor longus muscles at approximately the midportion of the thigh was a tumor which had been sectioned and measured approximately 8 by 6 by 5 cm. The tumor appeared well demarcated and was firm in consistency. The cut surfaces were whitish and presented a whorled appearance. It compressed the surrounding muscles and appeared to surround the femoral artery and veins.

Follow-Up.—The patient was doing fine, without evidence of recurrence and walking well with her prosthesis, at seven months after surgery.

Submitted Diagnoses.—Malignant schwannoma, 17; neurofibrosarcoma, 5; fibrosarcoma, 6; neurogenic sarcoma, 4; neurilemoma, 2; schwannoma (edema due to pregnancy), 1; myxosarcoma, 3; hemangiopericytoma, 1; sarcoma, unclassified, 2; neurofibroma, 2.

Dr. Stout: The tissue removed in this case is hard to evaluate. It consists of two fragments. In both

there is a tumor which shows peculiar characteristics of growth. These consist essentially of a fair number of thick walled vessels of capillary size with lumens and lining endothelial cells, but with tremendously thick walls composed of long spindle shaped and stringlike cells the long axes of which parallel the long axis of the capillary. These cells are in rows from 8 to 15 cells thick. There is no definite evidence that they are smooth muscle cells or schwannian in origin. Where these cells are lacking the tumor apparently becomes more vascular because the capillaries lacking the thickened walls lie closer together. An Ungewitter stain fails to show any axis cylinders save only in one area where there are three normal nerve twigs. The neurites (axis cylinders) in these are very palely stained so that they would be difficult to identify if they were buried in the more fibrous parts of the tumor.

At first glance one might suspect that the curving cords of spindle shaped cells are Schwann's cells and the structures plexiform neurofibromas. I do not think that they are, for the cells in each cord surround a capillary and I have never seen this in neurofibroma. This tumor has some of the characteristics of a hemangiopericytoma. The peculiar cells accompanying the vessels and thickening the walls are not smooth muscle cells nor apparently are they Schwann's cells, so that they must be either connective tissue cells or pericytes. It seems to me more probable that they are pericytes, since there is no tumor growth which has capillaries with enormously thick walls like this except vascular leiomyoma, mesenchymoma, and hemangiopericytoma. When one gets away from the capillaries and their tremendously thick sheaths of connective tissue fibers and spindle shaped cells, the tumor has more the general appearance of a differentiated fibromatosis infiltrating between fat cells. It would be possible, therefore, to think of this tumor as a fibromatosis with hemangiopericytomatous areas. I cannot tell from these sections whether the infiltrating fibromatosis or the thick walled capillaries predominate throughout the whole bulk of the tumor; therefore, I do not know which name to feature. Judging solely by these sections I would say that the vessels predominate, and for that reason I am calling it a hemangiopericytoma with fibromatosis. Although the thigh is one of the favorite sites for the development of metastasizing hemangiopericytomas, none that metastasized looked like this tumor.

CASE 18

Diagnosis.—Giant cell tumor of myositis ossificans of sacral region.

Contributor.—Capt. R. C. Schaffer, USAF Hospital, Lackland Air Force Base.

History.—A 24 year old white man was transferred from flight training into the hospital because of pain localized in the midsacral region. The pain was steady and aching and was accentuated by any flexion of the back. There was hypesthesia over the last three sacral dermatomes. The patient had experienced difficulty in urination and defecation during the preceding four weeks. There was no motor weakness of the lower extremities nor sensory disturbances except as mentioned previously. Myelograms and repeated roentgenograms of the lumbosacral area were negative. When a hematoma developed over the midportion of the sacrum, incision and drainage was attempted, with much blood welling from the base of the mass in the sacrum. A tumor weighing 135 Gm. was excised from contiguous muscle.

Submitted Diagnoses.—Myositis ossificans, 16; traumatic myositis ossificans with giant cell metaplasia, 3; ossifying hematoma, 2; giant cell tumor, 10; giant cell tumor (aponeurotic origin), 1; aneurysmal bone cyst, 3; osteogenic sarcoma, 4; miscellaneous diagnoses, 4.

Dr. Stout: Now the diagnoses: This caused me a lot of pain, and I imagine that it caused others pain to try to identify it. I am not sure what that giant cell tumor, aponeurotic origin means. Four thought that the lesion was malignant.

The surgeon's operative note indicated that the lesion was overlying the posterior sacrum and destroying the bone over the first, second, third, and fourth sacral segments. Prior to surgery the roentgenograms interpreted as showing no involvement of the bony sacrum. However, these films were not read by a radiologist. Postoperatively the films were restudied by the radiology staff, and they thought that the preoperative interpretation of negative bone findings in such a difficult area may not have been valid. However, they refused to state that the sacrum was involved. I think that they thought that one film showed some irregularity posteriorly over the sacrum. We don't have, unfortunately, a good operative note which told anything more about the exact degree of involvement of those sacral segments. We don't know whether it was just superficial destruction or whether it was a more important degree of destruction.

The seminar slide shows an encapsulated lesion. Outside of the capsule and attached to it is striated muscle. Inside the capsule is first a zone of very vascular, well differentiated osteoid. The trabeculae show calcification in some areas. This zone is thin and interdigitates on its internal aspect with a fibrous, vascular tissue containing a considerable number of multinucleated giant cells having the appearance of osteoclasts and, in some places, some more osteoid which is well differentiated. This tissue in its turn has adherent to its internal surface rather recent blood clot.

If we can accept all of the statements contained in the history, this is a growth which developed in the soft tissues posterior to the sacrum and probably not in the midline. The muscle is simply attached to the capsule and does not interdigitate with bone, which is not usual in myositis ossificans. I would say that the giant cell tumor appearance is also against

this diagnosis, although I am told it is possible to have a giant cell tumor of a nodule of myositis ossificans, and I have seen 1 case which conformed in appearance to this complex. The question of whether it is proper to consider this a possible example of the tumor which Geschickter and Copeland have labeled parosteal osteoma of bone is difficult to say, for we do not know its exact relationship with the periosteum of the sacrum. I have never felt that I understood this peculiar growth, which is supposed to have the histological character of a benign lesion yet actually is a malignant osteogenic sarcoma. In any event most of their cases had an obvious close relationship with a long bone, and the photomicrographs shown in their report usually have some area with malignant appearing cells as well as well differentiated osteoid or bone. I can think of them only as osteogenic sarcomas with the major portion of the growth extending outward from the periosteum and little or no evidence of bone involvement.

Recently Fine and I have been studying osteogenic sarcoma in the soft tissues. There are two variants—one is a genuine malignant osteogenic sarcoma which is just as malignant as the same tumor arising in bone. We could assemble 12 cases of this sort. In addition, there were 4 cases which were probably myositis ossificans but had areas of anaplasia suggesting a malignant change. Without exception clinically these were not malignant and should not be called osteogenic sarcomas.

In this present case there is nothing to suggest malignancy, and I think the lesion can be regarded as benign. I hesitate to call this unequivocally a myositis ossificans because it is encapsulated and bone trabeculae do not interdigitate with muscle fibers. Yet I cannot think of any other way of dealing with the fact of bone formation about the periphery. The giant cell portion can be called giant cell tumor or fibrous dysplasia of the bony portion. Obviously a hemorrhage has occurred which must account for the increase in size.

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Pressure Breathing Therapy In Chronic Pulmonary Disease

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Intermittent positive pressure and intermittent positive-negative pressure on either the oropharynx or the thorax by means of a mechanical device is a practical method of artificial ventilation. It has proved increasingly popular in the treatment of chronic pulmonary disease, to effect alveolar hypoventilation with its resulting complications, but undesirable side effects must be guarded against.

PRESSURE BREATHING implies the application of positive and/or negative pressure either on the oropharynx or the thorax by means of a mechanical device. This pressure can be applied either continuously or intermittently. Continuous pressure has been widely used in aircraft engineering (pressurized airplanes) and to a limited extent in clinical medicine. The application of intermittent pressure, however, presents itself as the most useful and practical method of artificial ventilation. Besides its use in the treatment of respiratory arrest or paralysis, it has become increasingly popular in the treatment of chronic pulmonary disease.[1, 14]

PURPOSE

The main purpose of pressure breathing in the management of patients with chronic pulmonary disease is to produce a beneficial effect upon alveolar hypoventilation. Alveolar hypoventilation results in a rise of alveolar carbon dioxide tension and a drop in alveolar oxygen tension and leads to anoxia, carbon dioxide retention, and finally, respiratory acidosis and damage of the respiratory center. In addition to its ventilatory ill effects, alveolar hypoventilation results in pulmonary hypertension and finally cor pulmonale. When the hypoventilation is due to bronchospastic disease, bronchodilators usually are sufficient to reverse its effects. However, when the bronchospasm is superimposed in patients with primary changes in the viscoelastic properties of the lung, little improvement may be noted after bronchodilator treatment. This is particularly true in some types of chronic pulmonary emphysema where the primary lesion lies in a loss of lung elasticity which leads to uneven ventilation of the lung. Under these conditions, the application of an additional transpulmonary pressure gradient promotes the ventilation of underventilated lung areas and restores the alveolar gas tensions to normal. In addition to the ventilatory improvement, the increased flow rates during expiration help to remove accumulated secretions. When combined with bronchodilator aerosols, the latter function becomes even more effective.

Dr. Maurice S. Segal, clinical professor of medicine, Tufts University School of Medicine, and director, Lung Station (Tufts) and Department of Inhalation Therapy, Boston City Hospital, presented this material as a guest speaker at the Texas Medical Association annual session, Galveston, in April, 1956. His co-authors are, respectively, clinical instructor and assistant professor in medicine assigned to the Lung Station and Department of Inhalation Therapy.

SIDE EFFECTS

Pressure breathing is a potent form of therapy with potential side effects when not properly applied. The application of excessive pressure differences may lead to hyperventilation, respiratory alkalosis, and tetany. Excessive positive pressures on the oropharynx may lead to overdistention of the lung, air leakage, and finally lung rupture. Overdistention in itself causes injury to the lung, the distention being related to time (duration of pressure), as well as to the actual pressure.

Positive pressure may be transmitted into the gastrointestinal tract and lead to distention of the stomach and intestines. This may produce undesirable vagal reflexes following the gastrointestinal dilation, increased difficulties of expanding the lungs by any means, and actual retardation of the venous return by a tense abdomen. Excessive negative pressures lead to vasodilation, pulmonary hemorrhage, and finally, acute cardiac dilatation. In normal subjects, the effects of positive oropharyngeal pressures correspond to the effects of negative pressures applied on the thorax either by tank or cuirass respirators.

The continuous application of pressure prevents the chest from coming to rest. During positive pressure breathing, the patient exerts his expiratory muscles in order to prevent overdistention, and during negative pressures, he exerts his inspiratory muscles in order to prevent collapse of his lung. This may lead to an actual increase in the work of breathing.

EFFECTS UPON CIRCULATION

In addition to the direct effects upon the pulmonary ventilation, one has to consider the effects upon the circulatory hemodynamics (fig. 1). During inspiration in normal breathing, the venous return to the right side of the heart is increased, while right ventricular and pulmonary arterial pressures fall though the pulse pressure increases. After the beginning of the expiratory phase, both right heart pressure and pulse pressure decrease markedly. The changes in the left heart are almost opposite. A reduction of the arterial and ventricular pressures at the onset of inspiration is followed by a rise during the latter phase of inspiration and early phase of expiration, and a subsequent fall during the greater part of active expiration. The right ventricular stroke volume is increased during inspiration and decreased during expiration. The left ventricular stroke volume is increased during expiration because of an increased return from the pulmonary circulation and becomes decreased during inspiration.

The inspiratory increase of pulmonary artery flow occurs despite a small increase in pulmonary bed resistance. The increase of resistance and of venous return leads to a larger residual volume in the right heart during inspiration. Part of the residual volume is ejected during expiration and expiratory pause.[8]

These hemodynamic changes occur in the normal subject but do not necessarily follow the same pattern in pulmonary disease. If pulmonary bed resistance, for instance, is already high, a further increase during inspiratory inflation of the lung could be so great that the increased venous return is unable to produce an increase in stroke volume if the heart muscle is damaged. The latter is frequently observed, independently, in patients with pulmonary disease.

In intermittent positive pressure breathing (IPPB) the same mechanism comes into play in reverse fashion, but the decrease of right heart output during rising mask pressure may be made up during the phase of falling mask pressure. The reverse occurs in the left heart but with limitations, because in-

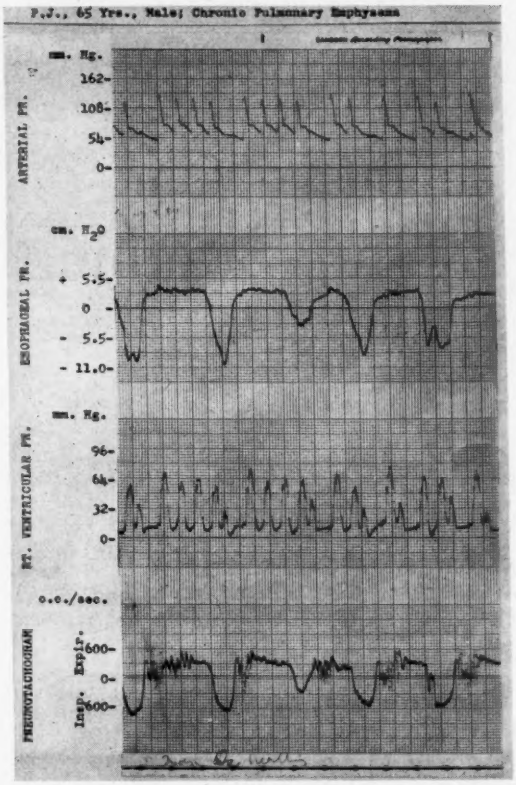

Fig. 1. Pressure changes in peripheral and pulmonary circulation with intrathoracic pressure changes in a patient with chronic pulmonary emphysema during oxygen breathing. Note the marked ball-valving in the expiratory flow pattern.

creased output during inspiration with IPPB can be maintained only during short periods (three to five beats) by displacement of pulmonary blood. If the inspiratory mask pressure is prolonged, the arterial pulse pressure decreases after the initial rise. Thus the variation in stroke volume in the right ventricle is the important part to consider in relation to changes in cardiac output.

The right ventricular net filling pressure during IPPB is decreased during periods of increased intrapleural pressure. The reverse occurs during periods of decreasing intrapleural pressure. Starling's law postulates that the deficit in cardiac output during

increased intrapleural pressure can be compensated for during the period of decrease in intrapleural pressure. If the expiratory time is sufficient, compensation may be complete.

Depending on the presence of normal circulating blood volume, existing vascular tone, and capacity of reflex vasoconstriction, the peripheral pressure rises

(1) gradual increase to a maximum of 25 cm. of water during inspiration; (2) rapid pressure drop early in expiration with mean pressure during expiration near to zero; (3) inspiratory time not to exceed expiratory time, thereby allowing as many heart beats to occur during expiration with increased stroke volume as during inspiration (fig. 2).

Maloney and others[9, 10] have stressed repeatedly the advantage of positive and negative pressure

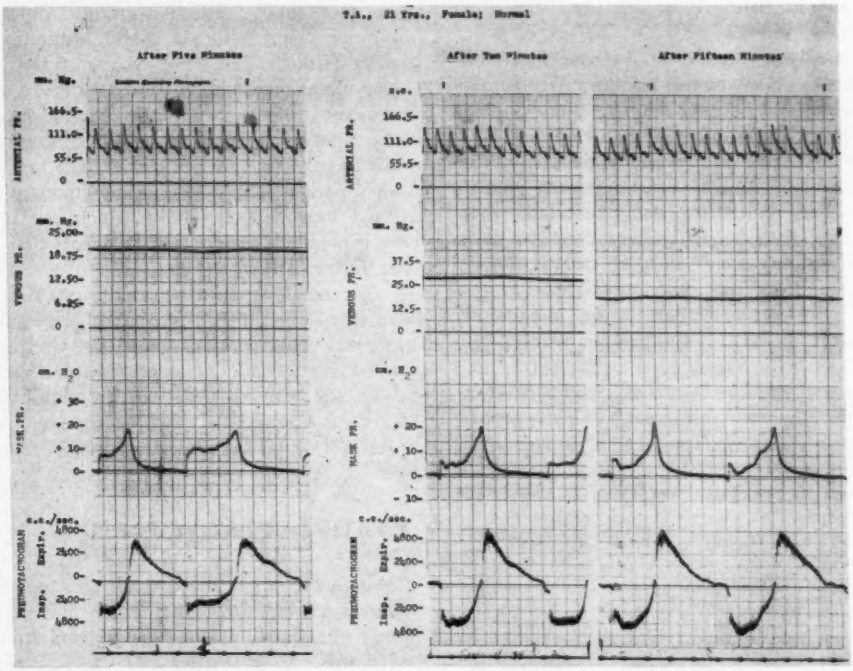

Fig. 2. Ventilatory and circulatory dynamics in a normal patient during the course of intermittent positive pressure breathing with pressure applied during inspiration by Bennett apparatus after 5 minutes, 10 minutes, and 15 minutes. Note the compensatory rise and consecutive drop in venous pressure and increased influence of respiration on arterial pressure.

with the rise of right auricular pressure (fig. 2). The increased peripheral pressure increases the capillary filtration pressure resulting in water loss to the tissues. It has been shown that positive pressure breathing for 30 minutes at 30 mm. of mercury causes a loss of 4 per cent of blood volume to the tissues. If sympathetic pathways are ineffective, the reconstitution of the venous gradient is greatly diminished or absent, and eventually a dangerous decrease in venous return, cardiac output, and blood pressure occurs. The same holds true for hyperactive sympathetic pathways where maximal reflex vasoconstriction has been utilized already.

In order to compensate for the decreased right ventricular output during the inspiratory phase of rising intrathoracic pressure, an ideal mask pressure with the following characteristics has been proposed:

breathing in the presence of respiratory failure and/or circulatory embarrassment, the IPPB-type machines causing a decrease in cardiac output which may be very serious.[9, 10] The clinician should keep this in mind in patients with chronic pulmonary emphysema in whom a diminished cardiac output type of failure may be present. These investigators also concluded that cardiac output and arterial blood pressure varied with the mean airway pressure during the respiratory cycle rather than with any particular wave form.

POSITIVE PRESSURE

The application of positive pressure to the airway during the inspiratory phase of respiration (with the use of a mask or mouthpiece), commonly referred

to as intermittent positive pressure breathing, may be abbreviated as IPPB/I.[7, 11, 13, 15, 17] The expiratory phase of respiration is entirely passive.

The devices employed to produce this form of therapy usually consist of an inspiratory demand valve operating on a flow sensitive or pressure sensitive principle and require the use of oxygen or oxygen-air mixtures. The more commonly employed are the Bennett, M.S.A., Emerson, Halliburton, and Monaghan valves. Positive pressures of 10 to 20 cm. of water are usually used and, on the whole, have been well tolerated. Therapeutic aerosols are generally administrated simultaneously during the inspiratory positive pressure phase of breathing.

This type of therapy has found its greatest usefulness in progressive bronchospastic disorders wherein uneven pulmonary ventilation exists, particularly when employed along with bronchodilator aerosols. It is especially helpful in patients with the carbon dioxide intoxication syndrome and respiratory acidosis.

It is generally assumed that during inspiration, positive pressure by mask is comparable to the negative pressure by body respirator and that during expiration, negative pressure by mask is comparable to positive pressure by body respirator.[18] This is probably true for normal lungs. However, in patients with changes of viscoelastic properties of the lungs, these pressures might not be transmitted, as elastic and coordinating structures have been upset by the uneven damaging of the intermediate tissues (fig. 3).

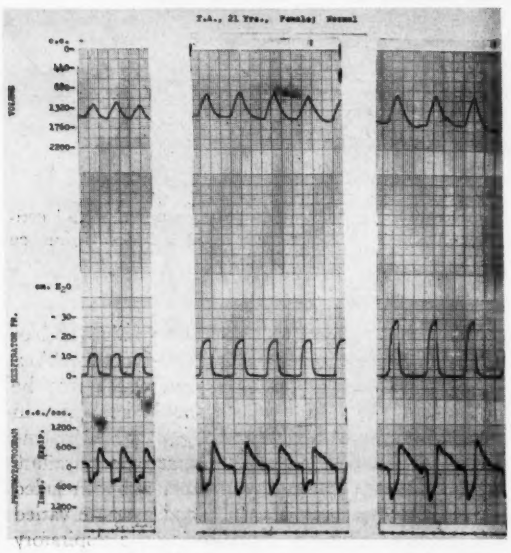

Fig. 3. Ventilatory dynamics in a normal patient with Emerson chest respirator at different pressure settings. The mean flow and tidal volume increase when the pressure gradient in the respirator is increased from 10 to 20 cm. of water. There is, however, no further increase in ventilation with a pressure gradient of 30 cm. of water. It will be noted that the respirator produces considerably lower flow rates than the intermittent positive pressure breathing in figure 2 with comparable pressure settings.

We would stress again that this form of pressure breathing should not be employed in patients under circulatory stress, for example, patients who are on the verge of or in peripheral vascular collapse, with heart failure, or having spinal anesthesia. If IPPB/I is used in the presence of high resistance to air flow, the peak mask pressure is attained quickly, and expiration starts before a useful inspiration has been accomplished. The rapid cycling of the pressure device under these circumstances indicates severe bronchospastic disease, and little can be expected from this type of treatment if the bronchoconstriction cannot be relieved by other means. The same holds true for a patient with a very low pulmonary compliance (stiff lung). The volume which can be delivered by a pressure setting may be insufficient to permit an adequate tidal volume.

In general, we do not use pressures above 15 to 20 cm. of water in the properly selected patient. In order to avoid undue fatigue, the sessions are limited to 15 to 20 minutes and are repeated three or four times daily. In patients with cardiac complications, this form of treatment is used with much caution, and pressures of 12 cm. of water are seldom exceeded.

POSITIVE-NEGATIVE PRESSURE

Inasmuch as low mean mask pressures produce in general less circulatory side effects, the addition of a phase of negative pressure during expiration has been advocated. Most IPPB/I machines are tripped into inspiration by an initial respiratory effort on the part of the patient, thus producing an initial small negative mask pressure. The latter is adjustable in the Halliburton unit. However, IPPB/I as well as intermittent positive and negative pressure breathing (IPNPB) machines and tank or cuirass respirators are regulated primarily by the pressure setting. The volume delivered by a given pressure setting depends upon the physical properties of the patient's lung and thorax. To be sure that a given pressure setting delivers an adequate ventilation, one has to measure minute ventilation, which occasionally may be quite cumbersome. Many failures in this form of treatment, especially in paralytic polio, are simply due to the fact that the patient is chronically underventilated. The ventilatory needs of a patient can be easily determined by a nomogram,[12] and adequate ventilation can be checked by determination of arterial blood gases and hydrogen ion concentration. Cherniack has compared the effects of mechanical exsufflation with the effects of a conventional tank respirator in patients with chronic pulmonary emphysema.[6] Because

the pressures of the conventional tank respirator were set in a fashion which did not allow delivery of adequate minute ventilation, there was no improvement with this form of therapy. On the other hand, an exsufflator (alternating positive and negative pressures) delivering a much larger minute ventilation, produced striking improvement in the pulmonary gas exchange.

It should be stressed again that the clinician using mechanical breathing devices must be certain that the patient is getting an adequate minute ventilation; otherwise the patient derives no benefit from the treatment and might actually get worse. The Engström respirator,[4, 5] which allows accurate regulation of minute ventilation instead of pressure regulation, has proven very successful in Europe where it has been used in the management of patients with poliomyelitis, tetanus, and chronic pulmonary disease.

A special form of this type of therapy has been introduced as exsufflation with negative pressure by Barach and his associates.[2, 3] During inspiration, a positive mask pressure is built up. This is changed over to a negative mask pressure during expiration in a fraction of a second (fig. 4). Because of the instantaneous development of a large transpulmonary pressure gradient, there are achieved high expiratory flow rates which may approach the flow rates of a successful cough. We have found this form of therapy with the multifunction Vent-El-Aire unit* very useful in patients with trapped bronchopulmonary secretions, bronchiectasis, lung abscess, and poliomyelitis.[13, 16] However, in patients in whom a change of the viscoelastic properties of the lungs has occurred (chronic pulmonary emphysema), the development of a large transpulmonary gradient frequently

**Manufactured by the Eliot Medical Plastic Co., Inc., Lynn, Mass.*

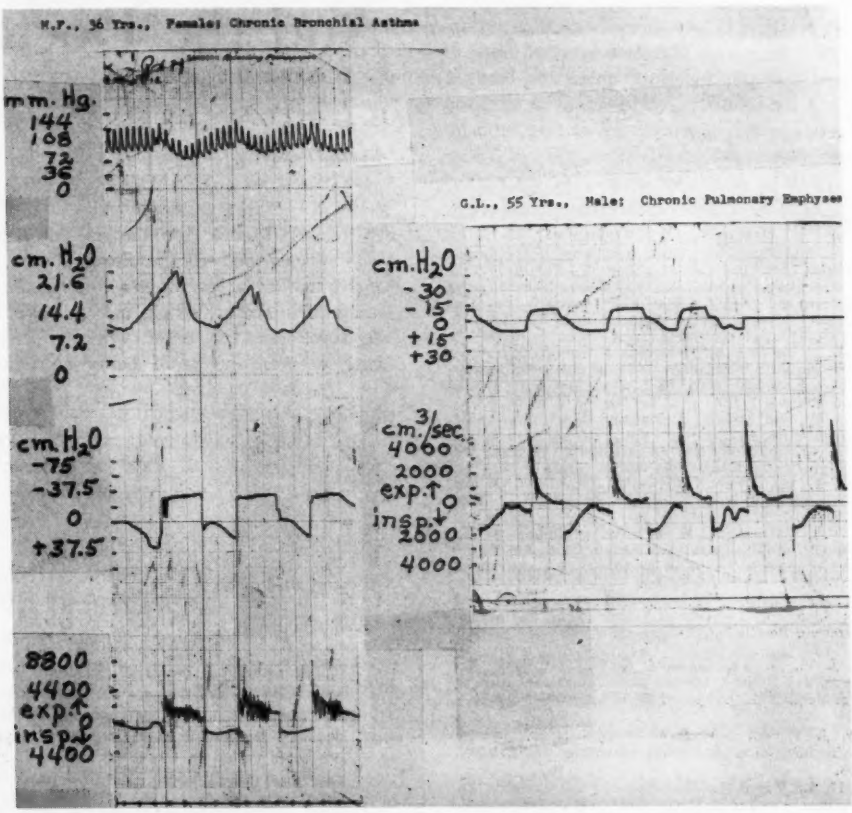

Fig. 4. Influence of exsufflation of pulmonary and circulatory dynamics in a patient with chronic bronchial asthma (left) and chronic pulmonary emphysema (right). Note the high initial expiratory peak flow rates with rapid fall (ball-valving) in the patient with chronic bronchial asthma. Inspired and expired volumes are comparable in this patient while in the patient with chronic pulmonary emphysema the expired volume is markedly less than the inspired volume. The large transpulmonary pressure gradient on the left tracing produces marked circulatory changes, but the flow pattern is not superior to the one on the right, where the pressure gradient is only one-third (Vent-El-Aire unit).

leads to expiratory ball valving or bronchial chattering. In spite of a momentary high expiratory air flow rate, the air trapping during expiration leads to overdistention of the lungs. The disturbances in expiration may be largely prevented in patients with a definite bronchospastic component by administering bronchodilator aerosols before or during positive and negative pressure therapy and may be minimized in the patients with lowered compliance (stiff lungs) by this technique. In these patients, we have observed comparably high expiratory flow rates with much larger expiratory volumes if IPPB/I with bronchodilators was employed.

SUMMARY

The effects of intermittent positive pressure breathing (IPPB/I) and intermittent positive-negative pressure breathing (IPNPB) on circulation and respiration have been recorded. The usefulness of both of these forms of therapy, their limitations and hazards, and equipment for them are described. The clinician must be certain that the patient is getting an adequate minute ventilation at all times.

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Drs. Segal, Goldstein, and Attinger, Boston City Hospital, 818 Harrison Avenue, Boston 18.

Industrial Accidents And the Doctor

H. C. PITTMAN, LL.B.

Austin, Texas

THE NUMBER of industrial accidents occurring in Texas and the resulting claims and controversies under our workmen's compensation laws continues to increase. When, in 1913, we were among the first states to enact workmen's compensation laws, we were far from being an industrial state.

H. C. Pittman is chairman of the Industrial Accident Board.

The number of industrial accidents in Texas, with resulting claims under workmen's compensation laws, continues to increase. As medical evidence is required in each case, more physicians are being called on to submit reports to the Industrial Accident Board. Here are some of the basic facts the doctor should know.

Now it has been estimated that Texas is third in the nation in industrially employed, exceeded only by New York and California, and it is predicted that Texas will be first by the turn of the century. As accidents are reported to the Industrial Accident

Board in increasing numbers, so are the number of claims and controversies being set for hearing increasing with the result that more and more physicians are being called upon to submit reports to this Board. The Board welcomes this opportunity to come before the doctors of Texas and present information about our workmen's compensation laws and procedure.

The Industrial, Accident Board office is located on the first floor of the Walton Building, Austin, where reports of injury are received at the rate of 950 per working day and claims for compensation filed at the rate of 250 per day. All hearings are held in Austin. There are three members of the Board, appointed by the Governor for six year terms. One member represents the employees, one the employer, and one is an attorney who serves as chairman of the Board. The volume of claims handled by the Board has more than doubled within the past 10 years. Last year there were 227,099 industrial accidents reported to the Board with a resulting 61,484 controverted claims considered by the Board. There has been no comparable increase in personnel of the Board, and its equipment and facilities are generally recognized as being pitifully inadequate. For example, under the present volume it takes 10 weeks to get a case on a docket and 8 days to answer a letter of inquiry. No statistical records are maintained on the types or causes of accidents, and no cross index system of any sort is established. More than half the controverted claims handled by the Board are settled by compromise settlement agreements before the Board. Almost 90 per cent of the final awards entered by the Board are appealed to a court of law. Last year the Board entered awards in 7,010 claims, notices of intent to appeal were received in 6,912 of these cases, and at least two-thirds of these appealed cases were settled by agreed judgment after suit was filed in the county where the injury occurred.

RULES AND PROCEDURES

In coping with the problems of administering our workmen's compensation laws under present conditions, the Board, under its rule making power, has adopted many administrative rules that are not commonly known by attorneys or doctors who do not regularly practice before the Board. It is estimated that attorneys represent claimants in approximately 20 per cent of the claims filed. Medical evidence is required in all cases. Attorneys' fees are limited by law to 15 per cent of the first $1,000 recovered before the Board and 10 per cent of each additional $1,000. The maximum fees increases to 33⅓ per cent of the amount recovered on appeal from the Board's action.

Hearings are held before the Board in Austin every other Tuesday, with approximately 500 cases set for each hearing date. The great majority of the cases are determined on the basis of written evidence, including narrative medical reports. All oral testimony given at these hearings is given under oath, and a transcript is available. Medical evidence is required from both parties in all cases set for hearing. Cases in which no medical evidence is filed by the claimant or his attorney are routinely cancelled to be reset only upon receipt of an up-to-date narrative medical report. This means that the case must again take its turn upon the docket. Board rules place the burden of supplying medical evidence upon the claimant and give the claimant the right to file medical evidence from a doctor of his choice or accept medical evidence filed by the insurance carrier.

The employer or insurance carrier has the right to designate the treating physician in case of industrial injury and also is liable for emergency first-aid treatment. If it be shown that the medical attention being provided is being furnished in "such a manner that there is reasonable ground for believing that the life, health or recovery of the employee is being endangered or impaired thereby," the workmen's compensation act authorizes the Board to order a change in the treating physician. If the employer has knowledge of the injury and refuses to furnish treatment, the employee has the right to select his own physician and ask the Board to order the payment by the insurance carrier of the expense of this treatment.

The insurance carrier has the right to have a claimant examined by a physician of its choice. Board rules require that request for such examination must show that the claimant has refused to submit to such an examination, must show the necessity for such an examination, and must be filed with the Board at least seven days before the case is set for hearing. The Board then has authority to order the claimant to report to the physician designated by the insurance carrier and to delay the entering of an award until the order is obeyed. In these instances the physician receives a copy of the Board's order and is required to file a copy of his report with the Board within 10 days after the examination is made.

Our workmen's compensation laws restrict the liability of the insurance carrier for "reasonable medical attention, nursing, or chiropractic services" to that authorized within 91 days from date of injury. The liability for hospital services is limited to that authorized within 180 days from date of injury. In actual practice, there are cases in which the insurance carrier voluntarily pays for medical attention and treatment beyond these periods; however, strict liability is limited to the 91 and 180 days periods. If the Board finds that there is a necessity for surgery, that

such surgery is not ordinarily unsafe, and that it will materially benefit the injured party, the Board has authority to order such surgery at the expense of the insurance carrier even though this order is entered outside the statutory 91 day period. When such an order is entered, a copy of the order is furnished the physician at the time it is entered.

The law on this point actually reads as follows:

> During the first 4 weeks of the injury, dating from the date of its infliction, the [insurance carrier] shall furnish reasonable medical aid, hospital services, nursing, chiropractic services and medicines. During the fourth or any subsequent week, upon application of the attending physician or chiropractor certifying the necessity therefor to the Board and to the [insurance carrier], the Board may authorize additional medical attention, nursing and chiropractic services not to exceed one (1) week, unless at the end of such additional week the attending physician or chiropractor shall certify to the necessity for another week of medical or chiropractic attention or nursing or so much thereof as may be needed, but in no event shall such services be authorized for a period longer than ninety-one (91) days from date of injury.

The Board is seldom called upon to enforce this proviso of requiring weekly authorization after the first four weeks of treatment in light of present day conditions; however, it is the law and occasionally the point will be raised on a disputed medical bill.

There also is a proviso in our laws with regard to fees and charges made for medical aid, hospital services, or medicines furnished to injured employees. This section provides that all charges shall be "fair and reasonable, shall be subject to the regulation of the Board and shall be limited to such charges as are reasonable *for similar treatment of injured persons of a like standard of living where such treatment is paid for by the injured person himself or someone acting for him. In determining what fees are reasonable, the Board may also consider the increased security of payment afforded by this law*" (emphasis added). The Board relies upon this section in determining the reasonableness of bills submitted for payment.

COMMON QUESTIONS

The Board receives many inquiries from physicians and hospitals throughout the state. Some of the most common ones and the Board's replies perhaps would be of interest to the medical profession:

Question: Who is entitled to copies of my medical report?

Answer: In our opinion this is determined by the principles of ethics of the medical profession and by the question of contract between doctor and patient. Our opinion is that the person employing the physician and paying for the examination or treatment is entitled to the report.

Q.: How do I determine whether the insurance company will accept liability and authorize my services?

A.: If you know which insurance company carries the workmen's compensation insurance for the employer of the injured party, you should get in contact with the local representative of the company for authorization. In many cases the employer can assist in this. If you do not know who the insurance carrier is, you can write the Board giving the name of the employer, and we will give the name of the insurance company.

Q.: When should I submit my bill to the Board?

A.: You should determine from the claimant the date of his hearing before this Board and submit to the Board an itemized statement of your services, together with your bill, showing the type of treatment rendered and necessity therefor, and that it was furnished within the 91 day period. When writing the Board concerning an unpaid bill, always give the full name of the injured employee, the Board number of his claim if possible, the date of his injury, and the name of his employer.

Q.: If the Board denies the claim of the injured party and denies my bill, what are my rights?

A.: You have the right to appeal the Board's award as to your medical bill to a court having jurisdiction. This is also true in the event the Board does not allow the full amount of your bill and you desire to appeal. Notice of intention to appeal from an award of the Board must be filed with the Board within 20 days from date of the award and then the appeal perfected by a suit filed in a court of law within another 20 days. You should consult your attorney in this regard. Either party has the right to appeal from a Board's award, and if the Board allows your bill as submitted and the insurance carrier serves notice of intention to appeal, you will be notified of this action.

Q.: I treated an injured worker and later found that his claim was heard and determined by the Board without my knowledge and without action being taken on my bill. What are my rights?

A.: You have the right to ask the Board to set the case for hearing on the question of your medical bill. The case will be set, all parties notified, and the Board will enter a formal award as in cases previously described.

Q.: What type of medical reports does the Board require?

A.: The Board requires narrative reports, signed by the physician, establishing a definite prognosis and percentage of disability, if any. Charts should accompany reports describing amputation. Expression of the degree of disability should be given in terms of the part or parts of the body affected and

should be limited to the disability resulting from the industrial injury in question. A review of Section 12, Article 8306, relating to compensation payable for injuries to specific members of the body would be helpful in understanding how the degree of disability should be expressed for purposes of calculating benefits due under the Texas workmen's compensation act. For those desiring more information concerning the payment of compensation for industrial injuries, pamphlet copies of the workmen's compensation act are available.

CONCLUSION

Of course any attempt to cover this broad field in a single article is impossible. Many problems are faced by the Board in attempting to administer the workmen's compensation laws of our state with the sudden growth in the number of accidents reported, not the least of which is the difficulty in giving prompt attention to the questions asked by the members of the medical profession as they serve in individual cases. The facilities and number of personnel available for processing claims filed are extremely limited.

Every indication points to a thorough study of this problem of statewide importance by the current session of the Legislature, and the Board seeks the assistance of physicians in bringing this problem to the attention of their legislators. Ask them to visit the Board and see firsthand what is going on.

The Board sincerely encourages any physician to make inquiry whenever it can be of assistance and assures the medical profession of its desire to serve doctors as fairly and as quickly as possible.

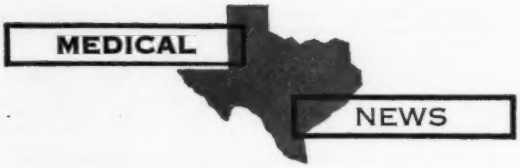
♦ Mr. Pittman, Industrial Accident Board, Walton Building, Austin.

Device Making TB Tests Safer

A device that makes testing sputum for tuberculosis germs in the hospital laboratory safer and easier has been developed by a Veterans Administration bacteriologist. It is an agitator for sputum specimens, made from a new kind of paint shaker by Abraham L. Rosenzweig at McGuire Veterans Administration Hospital in Richmond, Va., and the Veterans Administration has adopted the device as a standard item for its 173 hospitals.

US Health Expenses Surveyed

The American people spent more than $11.8 billion for personal health services during 1956, Health Information Foundation reports. More than 25 per cent of this sum, or almost $3 billion, was covered by voluntary health insurance.



★ Coming Meetings

Texas Medical Association, Dallas, April 27-May 1, 1957. Dr. Milford O. Rouse, Dallas, Pres.; Mr. C. Lincoln Williston, 1801 North Lamar Blvd., Austin, Executive Secy.

American Medical Association, New York, June 3-7, 1957. Dr. Dwight H. Murray, Napa, Calif., Pres.; Dr. George F. Lull, 535 North Dearborn, Chicago 10, Secy.

Current Meetings

APRIL

American College of Physicians, Boston, April 8-12, 1957. Dr. Walter L. Palmer, Chicago, Pres.; Mr. E. R. Loveland, 4200 Pine, Philadelphia 4, Secy.

American Dermatological Association, Belleair, Fla., April 13-17, 1957. Dr. Carroll S. Wright, Philadelphia, Pres.; Dr. J. Lamar Callaway, Duke Hospital, Durham, N. C., Secy.

American Proctologic Society, New Orleans, April 24-27. Dr. Rufus C. Alley, Lexington, Ky., Pres.; Dr. Karl Zimmerman, 3500 Fifth Ave., Pittsburgh 13, Secy.

Association of American Physicians and Surgeons, Delegates and Assembly, Miami Beach, Fla., April 25-27, 1957. Dr. Charles W. Pavey, Columbus, Ohio, Pres.; Mr. Harry E. Northam, 185 N. Wabash Ave., Chicago 1, Executive Secy.

Southwestern Surgical Congress, Wichita, Kan., April 15-17, 1957. Dr. John V. Goode, Dallas, Pres.; Dr. C. M. O'Leary, 207 Plaza Court Bldg., Oklahoma City, Secy.

Texas Air-Medics Association, Dallas, April 28-29, 1957. Dr. W. A. Ostendorf, Fort Worth, Pres.; Dr. C. F. Miller, P. O. Box 1338, Waco, Secy.

Texas Chapter, American College of Chest Physicians, Dallas, April 28, 1957. Dr. Walter C. Brown, Corpus Christi, Pres.; Dr. L. M. Sheffs, 510 Moore Bldg., San Antonio, Secy.

Texas Chapter, American Association of Public Health Physicians, Dallas, April 28, 1957. Dr. W. V. Bradshaw, Jr., Fort Worth, Pres.; Dr. A. R. Doane, 410 E. 5th, Austin, Secy.

Texas Dermatological Society, Dallas, April 28-29, 1957. Dr. Thomas L. Shields, Fort Worth, Pres.; Dr. E. N. Walsh, 1310 Medical Arts Bldg., Fort Worth, Secy.

Texas Diabetes Association, Dallas, April 28, 1957. Dr. Edwin L. Rippy, Pres.; Dr. Richard E. Nitschke, 1702 Nix Professional Bldg., San Antonio, Secy.

Texas Geriatrics Society, Dallas, April 30, 1957. Dr. Wendell D. Gingrich, Galveston, Pres.; Dr. J. O. S. Holt, Jr., 3707 Gaston Ave., Dallas, Secy.

Texas Heart Association, Dallas, April 28, 1957. Dr. D. D. Warren, Waco, Pres.; Mr. Edgar M. Brown, 404 Jesse H. Jones Library Bldg., Texas Medical Center, Houston 25, Executive Director.

Texas Industrial Medical Association, Dallas, April 30, 1957. Dr. V. C. Baird, Houston, Pres.; Dr. Robert A. Wise, Box 2180, Houston, Secy.

Texas Neuropsychiatric Association, Dallas, April 28, 1957. Dr. John L. Otto, Galveston, Pres.; Dr. Clarence S. Hoekstra, 8215 Westchester Drive, Dallas, Secy.

Texas Ophthalmological Association, Dallas, April 30, 1957. Dr. Thomas J. Vanzant, Houston, Pres.; Dr. Louis Daily, Medical Arts Bldg., Dallas, Secy.

Texas Orthopedic Association, Dallas, April 29, 1957. Dr. John J. Hinchey, San Antonio, Pres.; Dr. Margaret Watkins, 3629 Fairmount, Dallas, Secy.

Texas Society of Anesthesiologists, Dallas, April 28, 1957. Dr. Charles R. Allen, Galveston, Pres.; Dr. Randle J. Brady, 3317 Binz, Houston, Secy.

Texas Society of Gastroenterologists and Proctologists, Dallas, April 28, 1957. Dr. John McGivney, Galveston, Pres.; Dr. O. P. Griffin, 1101 Medical Arts Bldg., Fort Worth, Secy.

Texas Society of Pathologists, Dallas, April 30, 1957. Dr. Lloyd Hershberger, San Angelo, Pres.; Dr. Mervin H. Grossman, 3121 Bryan, Dallas, Secy.

Texas Society of Plastic Surgeons, April 27, 1957. Dr. Thomas D. Cronin, Houston, Pres.; Dr. Steve R. Lewis, University of Texas Medical Branch, Galveston, Secy.

Texas Surgical Society, Dallas, April 1-2, 1957. Dr. Truman G. Blocker, Galveston, Pres.; Dr. G. V. Brindley, Jr., Scott and White Clinic, Temple, Secy.

Texas Traumatic Surgical Society, Dallas, April 28, 1957. Dr. Russell Holt, El Paso, Acting Pres.;Dr. W. D. Marrs, 306 Broadway, Fort Worth, Secy.

Texas Tuberculosis Association, Fort Worth, April 4-6, 1957. Dr. Howard T. Barkley, Houston, Pres.; Mrs. J. V. Cooper, Waxahachie, Secy.

United States-Mexico Border Public Health Association, San Antonio, April 9-12, 1957. Dr. Guillermo Soberanes, Hermosillo, Sonora, Mexico, Pres.; Dr. Sidney B. Clark, 243 U. S. Court House, El Paso, Secy.

Seventh District Society, Austin, April 7, 1957. Dr. Leslie C. Colwell, Austin, Pres.; Dr. Robert N. Snider, 2410 Rio Grande, Austin, Secy.

Ninth District Society, Navasota, April 3, 1957. Dr. C. Marius Hansen, Navasota, Pres.; Dr. Lyman C. Blair, 1212 Rothwell, Houston, 2, Secy.

Eleventh District Society, Jacksonville, April 10, 1957. Dr. M. T. Braswell, Henderson, Pres.; Dr. Hugh F. Rives, Jacksonville, Secy.

State Tumor Conference, Wichita Falls, April 3, 1957. Mrs. Chester Robinsin, Wichita County Medical Society Tumor Clinic, Wichita Falls, Secy.

Texas State Board of Examiners in Basic Sciences, next examination, Austin, Galveston, and Houston, April 12-13. Henry B. Hardt, Ph.D., Fort Worth, Pres.; Mrs. Pearl A. Barrera, 407 Perry-Brooks Bldg., Austin, Chief Clerk.

MAY

American Association for Thoracic Surgery, Chicago, May 4-7, 1957. Dr. Cameron Haight, Ann Arbor, Mich., Pres.; Dr. Hiram T. Langston, 600 S. Kingshighway, St. Louis 10, Mo., Secy.

American Association of Genito-Urinary Surgeons, Hot Springs, Va., May 1-3, 1957. Dr. Archie L. Dean, New York, Pres.; Dr. John A. Taylor, 2 E. 54th St., New York 22, Secy.

American College of Chest Physicians, New York, May 29-June 2, 1957. Dr. Herman J. Moersch, Rochester, Minn., Pres.; Mr. Murray Kornfeld, 112 E. Chestnut, Chicago 11, Executive Secy.

American Gastro-Enterological Association, Colorado Springs, May 17-18, 1957. Dr. Sam A. Wilkinson, Jr., Boston, Pres.; Dr. H. Marvin Pollard, University Hospital, Ann Arbor, Mich., Secy.

American Gynecological Society, Hot Springs, Va., May 27-29, 1957. Dr. Norman F. Miller, Ann Arbor, Mich., Pres.; Dr. A. A. Marchetti, 3800 Reservoir Rd., N.W., Washington 7, D. C., Secy.

American Ophthalmological Society, Hot Springs, Va., May 30-June 1, 1957. Dr. Frederick C. Cordes, San Francisco, Pres.; Dr. M. C. Wheeler, 30 W. 59th, New York 19, Secy.

American Psychiatric Association, Chicago, May 13-17, 1957. Dr. Francis J. Braceland, Hartford 2, Conn., Pres.; Dr. William Malamud, 80 E. Concord, Boston 18, Secy.

American Surgical Association, Chicago, May 8-10, 1957. Dr. Loyal Davis, Chicago, Pres.; Dr. R. Kennedy Gilchrist, 59 East Madison, Chicago 3, Secy.

American Urological Association, Pittsburgh, May 6-9, 1957. Dr. George C. Prather, Brookline, Mass., Pres.; Dr. Samuel L. Raines, 188 S. Bellevue Blvd., Memphis, Tenn., Secy.

National Tuberculosis Association, Kansas City, May 5-10, 1957. Dr. Howard W. Bosworth, Los Angeles, Pres.; Mrs. Morrell DeReign, 1790 Broadway, New York 19, Secy.

Southwest Allergy Forum, Fort Worth, May 5-7, 1957. Dr. Truman C. Terrell, Fort Worth, Pres.; Dr. Sim Hulsey, 701 Fifth Ave., Fort Worth, Secy.

Southwestern Society of Nuclear Medicine, Houston, May 4-5, 1957. Dr. Henry H. Turner, Oklahoma City, Pres.; Dr. J. R. Maxfield, Jr., 311 Medical Arts Bldg., Dallas, Secy.

Texas Hospital Association, Houston, May 14-16, 1957. Mr. H. M. Cardwell, Lufkin, Pres.; Mr. O. Ray Hurst, Executive Director, 2208 Main, Dallas, Secy.

National and Regional

American Academy of Allergy, Philadelphia, Feb. 3-5, 1958. Dr. W. B. Sherman, New York, Pres.; Dr. Francis C. Lowell, 65 E. Newton, Boston, Secy.

American Academy of Dermatology and Syphilology, Chicago, Dec. 7-12, 1957. Dr. Nelson P. Anderson, 2007 Wilshire Blvd., Los Angeles 5, Pres.; Dr. James R. Webster, 55 E. Washington, Chicago 2, Secy.

American Academy of General Practice. Dr. J. S. De Tar, Milan, Mich., Pres.; Mr. Mac F. Cahal, Volker Blvd. at Brookside, Kansas City 12, Executive Secy.

American Academy of Obstetricians and Gynecologists, Los Angeles, April 21-23, 1958. Dr. R. Gordon Douglas, New York, Pres.; Dr. John C. Ullery, Ohio State University Hospital, Columbus, Secy.

American Academy of Ophthalmology and Otolaryngology, Chicago, Oct. 13-18, 1957. Dr. Erling W. Hansen, Minneapolis, Pres.; Dr. W. L. Benedict, 100 First Ave. Bldg., Rochester, Minn., Secy.

American Academy of Pediatrics, Chicago, Oct. 7-10, 1957. Dr. Edgar E. Martmer, Grosse Point 30, Mich., Pres.; Dr. E. H. Christopherson, 1801 Hinman Ave., Evanston, Ill., Secy.

American Association of Obstetricians and Gynecologists, Hot Springs, Va., Sept. 5-7, 1957. Dr. F. Bayard Carter, Durham, N. C., Pres.; Dr. E. Stewart Taylor, University of Colorado School of Medicine, 4200 E. 9th, Denver, Secy.

American Cancer Society, New York, Oct. 30-Nov. 1, 1957. Dr. D. A. Wood, San Francisco, Pres.; Mr. M. R. Runyon, 521 W. 57th, New York, Executive Vice-Pres.

American College of Allergists. Dr. Ethan Allen Brown, Boston, Pres.; Dr. Giles A. Koelsche, Mayo Clinic, Rochester, Minn., Secy.

American College of Gastroenterology, Boston, Oct. 20-23, 1957. Dr. Arthur A. Kirchner, Los Angeles, Pres.; Mr. Daniel Weiss, 33 West 60th, New York 23, Executive Director.

American College of Radiology, Feb. 7-8, 1958. Dr. Ira H. Lockwood, Kansas City, Pres.; Mr. W. C. Stronach, 20 N. Wacker Dr., Chicago 6, Executive Director.

American College of Surgeons, Atlantic City, Oct. 14-18, 1957. Dr. Daniel C. Elkin, Lancaster, Ky., Pres.; Dr. Michael L. Mason, 40 E. Erie, Chicago 11, Secy.

American Congress of Physical Medicine and Rehabilitation, Los Angeles, Sept. 8-13, 1957. Dr. A. B. C. Knudson, Washington, D. C., Pres.; Dr. Frances Baker, 1 Tilton Ave., San Mateo, Calif., Secy.

American Congress on Obstetrics and Gynecology, Los Angeles, April 21-23, 1958. Dr. R. Gordon Douglas, 116 S. Michigan Ave., Chicago 3, Chm.

American Heart Association, Chicago, Oct. 25-29, 1957. Dr. Edgar V. Allen, Rochester, Minn., Pres.; Mr. John D. Brundage, 44 E. 23rd, New York 10, Secy.

American Hospital Association, Atlantic City, Sept. 30-Oct. 4, 1957. Dr. Albert W. Snoke, New Haven, Conn., Pres.; Dr. Edwin L. Crosby, 18 E. Division, Chicago, Executive Director.

American Laryngological, Rhinological, and Otological Society, San Francisco, Calif., 1958. No 1957 meeting. Dr. Percy E. Ireland, Toronto, Ont., Canada, Pres.; Dr. C. S. Nash, 277 Alexander, Rochester 7, N. Y., Secy.

American Neurological Association, Atlantic City, June 17-19, 1957. Dr. H. Houston Merritt, New York 32, Pres.; Dr. Charles Rupp, 133 S. 36th, Philadelphia 4, Secy.

American Orthopedic Association, Hot Springs, Va., June 24-27. Dr. David M. Bosworth, New York, Pres.; Dr. Harold A. Safield, 715 Lake St., Oak Park, Ill.

American Pediatric Society, Carmel, Calif., June 17-19, 1957. Dr. Daniel C. Darrow, Mission, Kan., Pres.; Dr. A. C. McGuinness, 1427 I St., N.W., Washington 5, D. C., Secy.

American Public Health Association, Cleveland, Nov. 11-15, 1957. Dr. John W. Knutson, Washington, D. C., Pres.; Dr. R. M. Atwater, 1790 Broadway, New York 19, Executive Secy.

American Society of Anesthesiologists, Los Angeles, Oct. 14-19, 1957. Dr. Irving M. Pallin, Brooklyn, Pres.; Dr. J. E. Remlinger, Jr., 188 W. Randolph, Chicago 1, Secy.

American Society of Clinical Pathologists, New Orleans, Sept. 29-Oct. 4, 1957. Dr. J. L. Goforth, Dallas, Pres.; Dr. Clyde G. Culbertson, 1040-1232 W. Michigan, Indianapolis 7, Secy.

International College of Surgeons, U. S. Chapter, Chicago, Sept. 9-12, 1957. Dr. Curtice Rosser, Dallas, Pres.; Dr. Karl Meyer, 1516 Lake Shore Dr., Chicago, Secy.

Radiological Society of North America, Chicago, Nov. 17-22, 1957. Dr. C. Edgar Virden, Kansas City, Pres.; Dr. Donald S. Childs, 713 E. Genesee, Syracuse 2, N. Y., Secy.

Southern Medical Association, Miami Beach, Nov. 11-14, 1957. Dr. J. P. Culpepper, Jr., Hattiesburg, Miss., Pres.; Mr. V. O. Foster, 1020 Empire Bldg., Birmingham 3, Secy.

Southern Psychiatric Association, Nassau, Oct. 6-8, 1957. William H. McCullagh, Jacksonville, Fla., Pres.; Dr. Joseph L. Knapp, 210 N. Westmoreland, Dallas, Secy.

Southern Surgical Association, White Sulphur Springs, West Va., Dec. 10-12, 1957. Dr. Floyd W. McRae, Atlanta, Pres.; Dr. George G. Finney, 2947 St. Paul, Baltimore, Secy.

Southwest Regional Cancer Conference, Fort Worth, Sept. 20-21, 1957. Dr. O. L. Morphis, 815 Medical Arts Building, Fort Worth, Chm.

Southwestern Medical Association, El Paso, Oct. 9-11, 1957. Dr. Celso Stapp, El Paso, Pres.; Dr. Russell L. Deter, 1501 Arizona St., El Paso, Secy.

Tri-State Medical Assembly, Shreveport, September, 1957. Dr. H. O. Padgett, Marshall, Pres.; Dr. Jason Sanders, Sanders Clinic, Kings Highway, Shreveport, La., Secy.

State

Private Clinics and Hospitals Association of Texas. Dr. J. P. Anderson, Brady, Pres.; Mr. C. H. Rugeley, Wharton, Secy.

Texas Academy of General Practice, Dallas, Sept. 22-25, 1957. Dr. George V. Launey, Jr., Dallas, Pres.; Mr. Donald C. Jackson, 308 W. 15th, Austin, Executive Secy.

Texas Academy of Internal Medicine, Houston, December, 1957. Dr. Jay C. Crager, Beaumont, Pres.; Dr. Hugo Engelhardt, 1216 Main, Houston, Secy. Meetings restricted to members.

Texas Association of Blood Banks, New Orleans, 1957. Dr. C. D. Fitzwilliams, Fort Worth, Pres.; Miss Marjorie Saunders, 3707 Gaston Ave., Dallas, Secy.

Texas Association of Obstetricians and Gynecologists, Galveston, February, 1958. Dr. Arthur M. Faris, Houston, Pres.; Dr. Oran V. Prejean, 4317 Oak Lawn, Dallas, Secy.

Texas Club of Internists. Dr. Charles M. Darnall, Austin, Pres.; Dr. L. C. Carter, 2600 Procter, Port Arthur, Secy.

Texas Division, American Cancer Society, Austin, Dec. 5-6, 1957. Dr. Porter Brown, Fort Worth, Pres.; Mr. Curt W. Reimann, 5014 Bull Creek Rd., Austin 3, Executive Director.

Texas Pediatric Society, Austin, Oct. 17-19, 1957. Dr. E. M. Wier, Fort Worth, Pres.; Dr. James N. Walker, 5216 W. Freeway, Fort Worth, Secy.

Texas Proctologic Society, Fort Worth, February, 1958. Dr. Hugh Beaton, Fort Worth, Pres.; Dr. C. P. Hardwicke, 920 E. 32nd, Austin, Secy.

Texas Public Health Association, Dallas, Feb. 23-26, 1958. Mr. J. N. Murphy, Jr., Austin, Pres.; Mr. H. E. Drumwright, City Health Department, Dallas, Executive Secy.

Texas Radiological Society, San Antonio, Jan. 24-25, 1958. Dr. J. R. Riley, Corpus Christi, Pres.; Dr. J. E. Miller, 6407 Forest Lane, Dallas 30, Secy.

Texas Rheumatism Association, Houston, Dec. 6, 1957. Dr. Jesse W. Hofer, Houston, Pres.; Dr. Warren W. Moorman, 901 W. Leuda, Fort Worth, Secy.

Texas Society for Mental Health. Dr. Carmen Miller, Dallas, Pres.; Mr. John Lane, 2510 San Antonio, Austin, Executive Director.

Texas Society of Ophthalmology and Otolaryngology, Dallas, Dec. 6-7, 1957. Dr. Owen R. O'Neill, Paris, Pres.

Texas Urological Society. Dr. Jack Crow, Abilene, Pres.; Dr. Ian Thompson, John Sealy Hospital, Galveston, Secy.

District

First District Society, February, 1958. Dr. W. A. Jones, El Paso, Pres.; Dr. E. S. Crossett, 309 Medical Arts Bldg., El Paso, Secy.

Second District Society, Big Spring, 1957. Dr. Clyde Thomas, Jr., Big Spring, Pres.; Dr. Nell W. Sanders, P. O. Box 191, Big Spring, Secy.

Third District Society. Dr. A. G. Barsh, Lubbock, Pres.; Dr. William Klingensmith, 708 Monroe St., Amarillo, Secy.

Fourth District Society. Dr. Joe B. Stephens, Bangs, Pres.; Dr. S. H. Martin, 115 S. Park, San Angelo, Secy.

Fifth and Sixth Districts Society, Corpus Christi, July 5-6, 1957. Dr. James C. Sharp, Corpus Christi, Pres.; Dr. Winston E. Riley, 2400 Morgan, Corpus Christi, Secy.

Eighth District Society. Dr. E. Peter Garber, Galveston, Pres.; Dr. John Childers, Univ. of Texas Medical Branch, Galveston, Secy.

Tenth District Society, Port Arthur, Fall, 1957. Dr. Charles W. Castle, Liberty, Pres.; Dr. Richard O. Clements, 715 Main, Liberty, Secy.

Twelfth District Society, July 9, 1957. Dr. George C. Bryant, Waco, Pres.; Dr. J. T. Archer, Jr., Meridian, Secy.

Thirteenth District Society, Abilene, 1958. Dr. Roy Wilson, Seymour, Pres.; Dr. R. D. Moreton, 1217 W. Cannon, Fort Worth, Secy.

Fifteenth District Society. Dr. L. E. Rutledge, Daingerfield, Pres.; Dr. O. R. Taylor, Jr., Linden, Secy.

Clinics

Blackford Memorial Cancer Lectures, Denison, Nov. 5, 1957. Dr. John D. Gleckler, 211 N. Fannin, Denison, Chm.

Dallas Southern Clinical Society. Dr. Howard C. Coggeshall, Dallas, Pres.; Miss Helga Boyd, Medical Arts Bldg., Dallas 1, Executive Secy.

New Orleans Graduate Medical Assembly. Dr. Eugene H. Countiss, New Orleans, Pres.; Dr. Maurice E. St. Martin, 626 Maison Blanche Bldg., New Orleans 16, Secy.

North Texas-Southern Oklahoma Fall Clinical Conference, Wichita Falls, Oct. 19, 1957. Dr. Kenneth C. Bebb, 1518 10th, Wichita Falls, Chm.

Oklahoma City Clinical Society Conference, Oklahoma City, Oct. 28-30, 1957. Dr. Herman Fagin, Oklahoma City, Pres.; Miss Alma F. O'Donnell, 503 Medical Arts Bldg., Oklahoma City 2, Executive Secy.

Postgraduate Medical Assembly of South Texas, Houston, July 15-17, 1957. Dr. C. Forrest Jorns, 412 Jesse H. Jones Library, Houston 25, Secy.

International Medical Assembly of Southwest Texas, San Antonio, Jan. 27-29, 1958. Dr. John C. Parsons, 1125 Nix Professional Bldg., San Antonio, Secy.

Board Examinations

Texas State Board of Medical Examiners, Fort Worth, June 24-26, 1957. Dr. M. H. Crabb, 1714 Medical Arts Bldg., Fort Worth, Secy.

MEDICOLEGAL NOTES

Physician Not Guarantor

The statement that in the absence of a special contract, a physician or surgeon is never considered as warranting a cure or guaranteeing that his treatment will result in a benefit to his patient was made in the last issue of the *Journal*. Then I proceeded to discuss the problems of a special contract which might result in the physician or surgeon warranting or guaranteeing his services to the patient.

In this article will be considered some of the reasoning of the courts in holding that a physician or surgeon is not a warrantor or guarantor in the absence of a special contract.

This issue was first raised in Texas in 1858 in the case of Graham v. Gautier.* This case arose when the physician sued Graham for his fee for attendance upon the slaves of Graham. Graham admitted that the services had been rendered, but alleged that as a result of the unskillful and negligent treatment by Dr. Gautier, 10 slaves had died. Graham was asking for the value of the slaves who had died.

The testimony showed that the slaves were "taken sick in Matagorda County, with typhoid fever, and then the measles upon that." Because of the contagious nature of their sickness, they apparently were moved frequently and often housed in inadequate quarters. It seems from the testimony that the movement and selection of quarters was on the insistence of the owner of the slaves.

There seemed to be some conflict in the testimony as to the care and treatment given to the slaves by Dr. Gautier, but the court in its opinion made the following statement which forms the initial basis for the law in Texas today as to whether the physician or surgeon is to be considered a warrantor or guarantor:

> The duty and responsibility of a physician is well stated by a Judge in Pennsylvania: "It is a rule of law, that a medical practitioner *never insures the result*, but simply engages that he possesses a reasonable degree of skill, such as is ordinarily possessed by a profession generally, and to exercise that skill with reasonable care and diligence; and, again, to exercise his best judgment, but is not responsible for a mistake of judgment" (emphasis added).

This case sets forth the idea that the physician or surgeon never insures the results (although we have seen that such might be the case in the presence of a special contract), and the more recent cases substantiate the holding. Examples are such cases as Turner v. Stoker,† in which the court held that:

> The physician is not an insurer and does not warrant a cure, but he impliedly contracts that he possesses and he is required to use and exercise that degree of skill and care which physicians practicing in similar

*21 Tex. 111.

†289 S.W. 190.

locations ordinarily possess, but he does not impliedly warrant a cure and can be held as a guarantor only by virtue of an express agreement. Lee v. Moore, 162 S.W. 440; Ely v. Wilbur (N. J.), 10 A. 358.

Another case is Gifford v. Howell,‡ in which the court stated that:

> While the rule is that a *physician does not insure results,* he is required to have such reasonable skill as the profession generally possesses and to exercise such skill with reasonable care and diligence (emphasis added).

The question then arises as to what degree of care and skill is required of the physician by our courts. To some extent, the care and skill required is set out in some of the previously cited cases, but to point out better what measure is required of the physician, the following is quoted from the court's opinion in the case of Turner v. Stoker:†

> The family doctor is one of the most confidential persons the public has to deal with. His responsibility is one of the greatest of professions. When we are sick or injured, we place our life in his hands, and his advice and treatment may mean life or death. It is quite natural that he should be required to exercise that degree of skill, learning, and care which are ordinarily possessed and exercised by other such members of his profession in good standing in similar communities and under like or similar circumstances. He is not chargeable with negligence or failure to use his best skill and ability but only that care and skill exercised generally by physicians in same or similar communities.

The Court of Civil Appeals stated in Bowles v. Bourdon,§ which was subsequently affirmed by the Supreme Court of Texas, that:

> A physician's legal liability is measured by the fact that he merely engages that he has that reasonable degree of learning and skill which is possessed by others of his profession in the same locality, or by the average physician under the same or similar circumstances, and that he will exercise that skill and learning with reasonable care and diligence. In other words, exercising a high degree of care is not required of an attending physician; all that can be expected is the use of ordinary care, skill and diligence.

From the foregoing, it is clear that in the absence of a special contract, a physician is never considered as warranting a cure or guaranteeing that his treatment will result in benefit to the patient. Also, the quotations from the last two cited cases will give an indication of the care and skill that will be required of the physician by our courts.

—PHILIP R. OVERTON, Austin.

MEDICAL MEETINGS

Dentists Plan Pain Symposium

The American Academy of Dental Medicine will hold its eleventh annual meeting in Boston May 29-June 1. Outstanding clinicians will present various aspects of the nature and causation of pain arising in the oral cavity, in adjacent areas, and in the remainder of the body, and the program is planned to be of interest to the medical as well as the dental profession. Reservations and additional information can be obtained from Dr. Arthur Gold, convention chairman, 26 Maple Street, Springfield 3, Mass.

‡*119 S.W. (2d) 578.*
§*213 S.W. (2d) 713.*

Pan American Cancer Congress Ahead

The United States Department of State has invited the ministers of health of the 21 nations of the western hemisphere to the First Pan American Cancer Cytology Congress, to be held April 25-29, in Miami Beach, Fla. It will also be of interest to American researchers, medical practitioners, specialists, public health workers, and educators, who are invited to attend.

The Congress will present the latest advances in cancer detection, in microbiology, radioisotopes, electron microscopy, and in the biology of cancer. Five clinical and cytology sessions and four basic science sessions will be included in the program. Climax of the session will be presentation of the $1,000 Wien Awards to scientists making most outstanding contributions to cancer cytology during 1956-1957.

Sponsors are the Cancer Cytology Foundation of America, Inc., Cancer Institute at Miami, Southern Society of Cancer Cytology, and University of Miami.

Registration fee is $15 with no fee for attending wives. Further information may be received from Dr. J. Ernest Ayre, President, in care of the Cancer Institute at Miami.

Student AMA to Meet May 3-5

More than 3,000 medical students are expected to attend the seventh annual meeting of the Student American Medical Association to be held May 3, 4, and 5 in Philadelphia.

Secretary of Health, Education, and Welfare Marion B. Folsom is the top speaker for the program, which includes a scientific presentation by Dr. C. P. Rhoads, cancer researchist from the Sloan-Kettering Institute, New York. A panel on "General Practice—Specialty; Trends in Practice" will include Dr. Kenneth Babcock, director, Commission on Accreditation, Chicago; Dr. J. S. De Tar, president, American Academy of General Practice, Milan, Mich.; Dr. I. S. Ravdin, chairman, Board of Regents, American College of Surgeons, Philadelphia; and Dr. Edward L. Turner, secretary, Council on Medical Education and Hospitals, American Medical Association, Chicago. Past AMA President, Dr. Elmer Hess, Erie, Pa., will serve as moderator.

The introduction of student research papers is an addition to the Philadelphia meeting.

Nuclear Medicine Society to Meet

The second annual meeting of the Souhtwestern Society of Nuclear Medicine will be held May 4-5 in Houston. Practical demonstrations of radioisotope techniques; panel discussions on radioisotopes versus chemotherapy in blood dyscrasias and in malignant effusions, federal and state radiation protection codes, and pulse height discrimination technique; and papers covering a variety of topics are scheduled.

Details may be had from Dr. Herbert C. Allen, Jr., program chairman, Radioisotope Department, Methodist Hospital, Houston 25.

Goiter Specialists to Meet

Papers and discussions dealing with the physiology and diseases of the thyroid gland will constitute the program when the American Goiter Association holds its annual session May 28-30 in New York City. Further information may be obtained from the secretary, Dr. John C. McClintock, 149½ Washington Avenue, Albany 10, N. Y.

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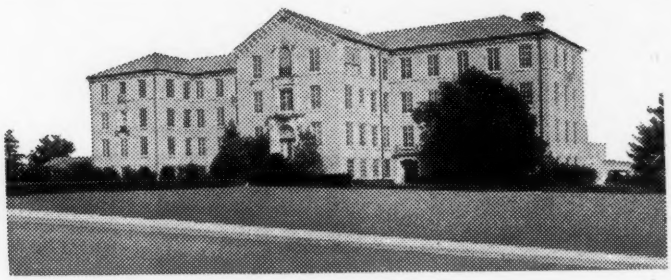
James K. Peden, M.D., Associate Psychiatrist
Ward G. Dixon, M.D., Associate Psychiatrist
Jerry M. Lewis, M.D., Associate Psychiatrist
LeeOwen S. Buford, M.D., Associate Psychiatrist
C. L. Jackson, M.D., Resident Psychiatrist

Mrs. Frances Campbell, R.N., B.S., Director of Nurses
Ralph M. Barnette, Jr., Business Manager
Miss Geraldine Skinner, Director of Occupational Therapy



WICHITA FALLS CLINIC-HOSPITAL

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James T. Lee, M.D.
Charles H. Wilson, M.D., F.A.C.S.
ORTHOPEDIC SURGERY
Jack E. Maxfield, M.D., F.A.C.S.
Clyde W. Parsons, M.D.
ORAL SURGERY
R. A. Mitchell, D.D.S., F.A.C.D.
E. E. Stewart, D.D.S.
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Julian H. Acker, M.D.

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Warren T. Kable, M.D.
Frank J. Lee, M.D.
ADMINISTRATIVE OFFICER
Bertha McNeill, Administrator

EDUCATION

Postgraduate Courses

Management of Common Dermatologic Problems, Houston, May 3-31.—Given by the University of Texas Postgraduate School of Medicine and Baylor University College of Medicine, this is a five weeks course to be held each Friday night from 7 to 9 p. m., beginning May 3 and ending May 31. The place will be M. D. Anderson Hospital and Tumor Institute, the tuition fee $10, the credit hours 10. Applications are available from the University of Texas Postgraduate School of Medicine, Texas Medical Center, Houston 25.

Pediatric Symposium, Houston, May 8-11.—Baylor University College of Medicine's Department of Pediatrics and the Division of Maternal and Child Health of the State Department of Health are sponsoring this symposium at the Texas Children's Hospital. Seven out-of-state physicians will speak. Details are available from Dr. Russell J. Blattner, Chairman, Department of Pediatrics, Baylor University College of Medicine, Houston.

Gastroenterology, Denver, May 13-15.—A three-day postgraduate course presented at the University of Colorado School of Medicine, the series will offer a broad and intensive review of present day concepts and pertinent recent developments in the field. The faculty will include 28 nationally-known guest physicians, a fact made possible by cooperation of the American Gastroenterological Association, which will hold its annual session in Colorado Springs immediately following the course. For details, write the Office of Postgraduate Medical Education, University of Colorado Medical Center, 4200 East Ninth Avenue, Denver 20.

Pediatric Advances for Pediatricians and General Practitioners, Philadelphia, May 27-31.—This course will be conducted by the staff of the Children's Hospital of Philadelphia, in collaboration with the Department of Pediatrics of the University of Pennsylvania and the staff of the Camden Municipal Hospital. Tuition is $110. Dr. Irving J. Wolman, Children's Hospital, 1740 Bainbridge Street, Philadelphia 46, can provide further details.

Practical Pediatric Hematology, Philadelphia, June 3-5.—The short refresher course will be conducted by Dr. Irving J. Wolman and other members of the Hematology Department, Children's Hospital, Philadelphia, under auspices of the Graduate School of Medicine, University of Pennsylvania. Tuition is $75. Dr. Wolman, 1740 Bainbridge Street, Philadelphia 46, can give further information.

Blood Group Incompatibilities and Erythroblastosis Fetalis, Philadelphia, June 6-7.—Dr. Neva Abelson and Dr. Thomas R. Boggs, Jr., of the Philadelphia Serum Exchange of the Children's Hospital, will conduct the course, also being presented under auspices of the Graduate School of Medicine, University of Pennsylvania. Tuition is $50, and details may be received from Dr. Irving J. Wolman, Children's Hospital, 1740 Bainbridge Street, Philadelphia 46.

Post Graduate Symposium on the Basic Sciences Related to Anesthesiology, Pittsburgh, June 10-14.—Presented by the University of Pittsburgh School of Medicine's Section on Anesthesiology, in cooperation with the Departments of Anesthesiology of the St. Francis, Allegheny General, Mercy, and Medical Center Hospitals, this course will be limited to 50 participants. Registration fee is $25. Full particulars should be obtained from: Chairman of the Committee on Graduate Medical Education, University of Pittsburgh School of Medicine, 3941 O'Hara Street, Pittsburgh 13.

Management of Chronic Kidney Disease, New York, June 24-25.—New York University Post-Graduate Medical School will present this full-time course under direction of Dr. Lawrence G. Wesson. It will deal with selection and interpretation of clinically available tests for the estimation of renal function. Advantages and disadvantages of each test will be considered. For details, write to the office of the Associate Dean of the University, 550 First Avenue, New York 16.

Management of Hypertension, New York, June 26-27.—Under the direction of Dr. J. Marion Bryant, this course will be concerned with the presentation, with appropriate clinical demonstrations, of the following subjects: brief summary of etiologic theories, psychological factors, the natural course, relative significance of blood pressure levels, eye grounds, cardiac silhouette, electrocardiogram, renal function, significance of obesity and arteriosclerosis, and unilateral renal disease. Emphasis will be placed upon use of hypotensive drugs. Other therapeutic measures, such as sympathectomy, nephrectomy, and diet therapy will be reviewed. Further information may be received from the Associate Dean, New York University Post-Graduate Medical School, 550 First Avenue, New York 16.

Occupational Medicine, New York, September 16-November 8.—Designed for physicians engaged in the practice of medicine in industry, this course is a full-time eight-week one offered by the New York Post-Graduate Medical School. Course content will cover preventive medicine, administrative medicine, occupational diseases, industrial hygiene. It is aimed at meeting the need for specialized training in industrial medicine. Instruction will be supplemented with field trips to industrial plants, governmental agencies, and union health centers, and opportunity will be given to attend medical, surgical, and clinical-pathological conferences in the New York University-Bellevue Medical Center. More information is available from the Associate Dean of the School, 550 First Avenue, New York 16.

UT Names Committee of 75

As the University of Texas prepares to observe its seventy-fifth year of service to the state, the Board of Regents designated 1958 for the determining of a broad outline of University development for the next 25 years.

Major responsibility for charting the objectives has been accepted by "The Committee of 75," which represents all state senatorial districts and a cross-section of Texas interests. Meeting for the first time in Austin on April 13, the group included: Dr. L. S. Oates, Center; Dr. Robert W. Kimbro, Cleburne; Dr. Kleberg Eckhardt, Corpus Christi; Dr. Herbert F. Poyner and Dr. William Seybold, Houston; and Dr. H. Frank Connally, Jr., Waco.

University of Texas Medical Branch

Dr. Wendell M. Stanley, Nobel Laureate from the University of California, and also professor of biochemistry and director of the virus laboratory of the University of California in Berkeley, gave the Meyer Bodansky Memorial Lecture at the University of Texas Medical Branch, Galveston, on March 11.

Dr. Michio Okamoto, professor of anatomy, Kobe Medical College and Mie Prefecturae University, Japan, spoke at the Medical Branch April 1 on "Recent Observations on the Cultivation of the Reticular Formation."

Dr. Sam L. Clark, Jr., assistant professor of anatomy, Washington University, St. Louis, also was a recent guest.

Another recent guest in Galveston was Col. Arthur B. Tarrow, chief of the anesthetization and operating section, Lackland Air Force Base, San Antonio.

Physical Medicine Prize Announced

To stimulate interest in the field, the American Congress of Physical Medicine and Rehabilitation will award annually a prize for an essay on any subject relating to physical medicine and rehabilitation. The contest, while open to anyone, is primarily directed to medical students, interns, residents, graduate students in the preclinical sciences, and graduate students in physical science and rehabilitation.

For further details of the competition, which closes June 1, query the American Congress of Physical Medicine and Rehabilitation, 30 North Michigan Avenue, Chicago 2.

From Rockefeller: Allergy Fellowships

The Board of Trustees, American Foundation for Allergic Diseases, announces availability of three fellowships in research and clinical allergy for a period of two years each, carrying a stipend of $4,500 the first year, $4,750 the second, and a total of $750 for laboratory and travel expenses during the period. Funds have been made available by John D. Rockefeller Jr. in a grant to the foundation.

Applications must be received by May 10, and notification of action will be sent to the candidate by June 10. Further information is available from Dr. Robert A. Cooke, chairman, Scientific and Educational Council, American Foundation for Allergic Diseases, 274 Madison Avenue, New York 16.

New York-Bellevue Physical Medicine Residencies

Approved residencies in physical medicine and rehabilitation are available at New York University-Bellevue Medical Center beginning July 1. American graduates with approved internships are eligible for OVR Fellowship, starting at $3,400 per year with added dependency allotment. Immediate application should be made to Dr. Joseph G. Benton, Institute of Physical Medicine and Rehabilitation, 400 East 34th Street, New York 16.

Ophthalmology Fellowships Available

Six additional fellowships for residents in ophthalmology, to begin July 1, have been announced by the Guild of Prescription Opticians of America, Inc. Each is for $1,800, payable in monthly stipends over the three-year residency period. Applications must be in by May 15. Forms and information may be received by writing to Fellowships, Guild of Prescription Opticians of America, Inc., 110 East 23rd Street, New York 10.

Chest Physicians Slate New York Conference

Formal presentations, symposiums, round-table luncheon discussions, seminars, and motion pictures will be features of the twenty-third annual meeting of the American College of Chest Physicians, to be held May 29-June 2 at the Hotel Commodore in New York City. Fireside Conferences, begun in 1955, have proved so popular that they will be repeated, with 50 experts leading discussions. Copies of the program may be obtained from the college officers, 112 East Chestnut, Chicago 11.

Texas and Nation Note Medical Education Week

Medical societies in Texas and across the nation are joining in the second annual observance of Medical Education Week April 21-27, when the impressive story of the accomplishments of United States medical schools is told.

Purpose of the observance is to focus the attention of the American people on the national importance and indispensability of medical education. Its national sponsors are the Association of American Medical Colleges, American Medical Association, American Medical Education Foundation, National Fund for Medical Education, Student American Medical Association, and Woman's Auxiliary to the American Medical Association.

In Texas, the Dallas County Medical Society is participating in a variety of ways. Two special medical sections will be published by Dallas newspapers on Sunday, April 21. There will be a proclamation by the mayor of the city, radio and television time has been arranged for, service clubs will be addressed by medical speakers, and the *Dallas Medical Journal* carried a pertinent editorial in its March issue. Chairman of the observance there is Dr. M. T. Jenkins.

Cancer Foundation Gift

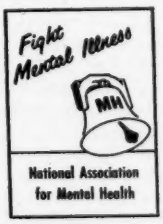
The University of Texas has received a $100,000 gift from the M. D. Anderson Foundation for the University Cancer Foundation. The Cancer Foundation operates at the M. D. Anderson Hospital and Tumor Institute, a component unit of the University, and is dedicated generally to strengthening the hospital's educational and research program.

This gift represents the first of five annual payments under a grant of $500,000 from the Anderson Foundation, one of the hospital's chief benefactors.

OF GENERAL INTEREST

Doctors, Drugs Gaining On Mental Illness

More than 16,000,000 Americans—1 in every 10—are suffering from a mental or emotional disorder, a study recently concluded by the Commission on Chronic Illness reveals. One out of every 10 children born each year will need to go to a mental hospital sometime during his life-



time, and there are more people (175,000) in mental hospitals right now than in all other hospitals combined.

Of the 20,000,000 patients in general hospitals last year for physical ailments, about 6,000,000 had illnesses caused by emotional disturbances, and mental disorder is an impor-

tant factor in 50 to 70 per cent of the medical cases treated by physicians.

Yet—the chances of recovery today are far greater than they were just a few years ago, recent statistics show. For instance, schizophrenic patients have at least a 60 per cent chance of partial or total recovery providing they get early and adequate treatment. Involutional psychosis and manic-depressive psychosis (mental disorders of middle age) show 65 and 75 per cent recovery rates respectively. In a well-staffed, well-equipped mental hospital, 70 per cent of patients improve and recover, although in the average hospital the rate goes down to 45 per cent.

For the first time, during 1955, evidence pointed to the likelihood of a break in the steep 25-year climb of the number of mental patients. Several state hospitals report that the discharge of mental patients has caught up with, or even exceeded, new admissions. During 1955, the number of resident patients increased by only 6,000 as compared to an average annual rise of 12,000 in the previous 10 years.

Beginning with Mental Health Week, April 28-May 4, and continuing throughout the month of May, workers for the National Association for Mental Health will be ringing doorbells throughout the country for voluntary contributions. Money given to the association is used to finance personal services for mental patients and their families, support research, establish community clinics, train personnel, support campaigns for better mental hospitals, and educate the public on the prevention of mental illness. Except for a very small percentage, all the money raised remains in the states and communities where it is obtained for the implementation of their programs.

Blood Banking Receives Current Consideration

Blood banks, their services and problems, are receiving wide consideration on local, regional, and national levels in early 1957, as the Joint Blood Council and allied groups strive to organize facilities and do research on the subject of blood banking for peace and war.

Equipped with a $50,000 Public Health Service grant, the Joint Blood Council on March 1 launched its nationwide survey of blood transfusion services. The two-year census has as its objectives the preparation of standards for accreditation of blood banks, development of a glossary of terms, inventory of research in blood and blood derivatives, and the assembling and analysis of needed data. As a preliminary to the main study, the council conducted a postcard survey of some 10,000 hospitals, clinics, and other groups asking, among other things, whether blood was obtained from the Red Cross, a community agency, or some other facility.

Conceived as a policy-deliberating, not operating body, the Joint Blood Council has five member organizations—American Medical Association, American Hospital Association, American National Red Cross, American Society of Clinical Pathologists, and American Association of Blood Banks. Its purpose is to stimulate research in blood and blood banking and to coordinate nationally efforts in this field.

In December, 1956, 26 federally licensed blood banks which are institutional members of the AABB, indicated readiness to participate in, and volunteered to collect 211,748 pints of blood over a 14-month period for, a project planned jointly last fall by the Federal Civil Defense Administration and the Red Cross: collection of fresh blood to be converted into units of serum albumin.

Activities of the AABB in the national scope resulting from its annual meeting in Boston, Mass., last fall include: (1) setting up of a certification and training program for blood banking technologists after January 1, 1960, plus a "grandfather clause" for participation of those already in the field; (2) designation of blood banks which may act in the capacity of training schools for technologists, through cooperation with the Registry for Technologists; (3) development of some special publications devoted to blood banking and planning of others; (4) development of a set of minimal standards for blood transfusion by the Committee on Standards of the AABB; (5) designation of regional reference laboratories available on a voluntary basis to cope with two major blood banking problems, the identification of unusual antibodies against red blood cells capable of causing incompatible transfusions and procurement of unusual blood types for special cases; (6) operation of a National Clearing House for the exchange of blood and blood credits, now with five district offices in San Francisco, Chicago, Dallas, Jacksonville, and New York.

Of regional significance is a decision of the Texas Association of Blood Banks at its November 30, 1956, annual session in Dallas to expand this association into a South Central Association of Blood Banks to include the states of Oklahoma, Arkansas, Mississippi, Louisiana, and New Mexico. These, with Texas, make up the South Central District of the National Clearing House program for blood banks.

During 1956, the Texas Association of Blood Banks Clearing House became a solvent operation, handled more than 12,000 transactions with blood credits and exchange, and continued to grow. Expansion of the association will open the way to doubling the number of participants in the clearing house, increasing official transactions substantially, and to increased efficiency and effectiveness of the entire operation.

Dr. E. E. Muirhead, Dallas, representing the Committee on Blood Banks at a meeting of the Texas Medical Association Executive Council in January, 1957, presented a resolution, which was adopted, reiterating that "it is highly desirable that matters dealing with blood banking, particularly of national scope, be considered and developed in conjunction with medical doctors active in organized medicine who by experience and practice are also active in the field of blood banking."

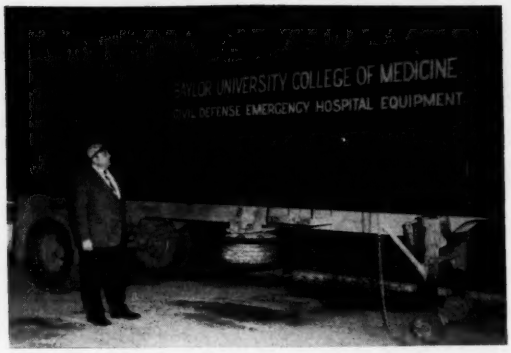
Wrong People Are Bearing Indigent Care Burden, Snoke Says

The self-paying patient in some hospitals is being unfairly taxed over the cost of his own care to help finance health personnel education and hospital care for the indigent and medically indigent, says Dr. Albert W. Snoke, president of the American Hospital Association, in the March 1 issue of *Hospitals*.

Paying patients and hospital employees are both shouldering some of the burden unfairly, he asserts, the latter by getting a lower wage than that received by their counterparts in the community who come to the hospital and get a lower hospital charge at the expense of the hospital employee.

Care for the indigent should be met by tax funds, Dr. Snoke believes. He advocates that hospitals collectively and individually should (1) receive current full cost from government agencies for both inpatient and outpatient welfare care; (2) receive supplemental funds from society in general; (3) receive current full cost from third-party agencies such as Blue Cross and other insurance agencies; and (4) spread cost for educational purposes over the entire population with emphasis upon those health groups that profit in the product without sharing in the expense of work.

Operation



Dr. Ralph E. Gray, Lake Jackson, chairman of the Texas Medical Association's Committee on National Emergency Medical Service, looks at a van containing equipment and supplies for a 200 bed emergency hospital. This hospital was set up in West Columbia; a second was set up in Arcadia.

"H-Bomb War Begins—Houstonians Flee Homes As City Threatened."

So read the headlines of a special newspaper issued to introduce—with "closed television" documentation and short-wave radio alerts—"Operation Rebound." An estimated 2,000 persons voluntarily participated in this first field trial by civilians of a 200 bed emergency hospital unit designed and owned by the Federal Civil Defense Administration, the trial being held March 29 in Harris, Brazoria, and Galveston Counties with Matagorda and Wharton Counties assisting.

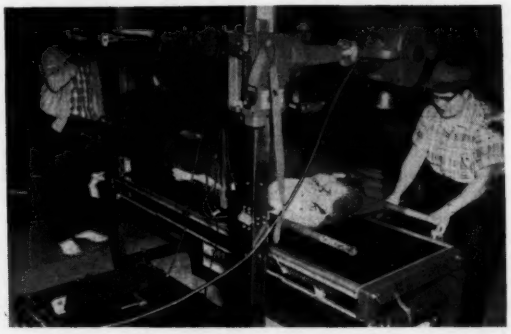
About 185 persons met in conference during the day at Baylor University College of Medicine, Houston, to hear a realistic discussion of public health problems—radiation, communicable disease, biological warfare, and chemical warfare—that might arise in case of disaster. This discussion by United States Public Health Service experts brought in from over the nation was followed by presentation of medical care problems to be anticipated, particularly with use of the emergency hospital unit to be tested.

Following appropriate alerts and with constant radio communication between key points, the field trial got under way during the afternoon with removal of two emergency hospital units in their individual vans from Houston to Arcadia and West Columbia, respectively. By nightfall, medical student evacuees with lifelike plastic-and-paint injuries began arriving at advanced aid stations and then were sent on to the emergency hospitals in West Columbia and Arcadia. There, teams of doctors, nurses, and allied personnel, with the help of Civil Defense communications operators, Boy Scouts, and other volunteers, took over the task of sorting, diagnosing, and treating the casualties. Senior medical students worked with the doctors, making the operation an educational effort as well as a trial of the hospital equipment and supplies. The activities continued until 6 a. m. Red Cross canteens in each hospital supplied food and drink throughout the procedure.

Baylor University College of Medicine and the University of Texas Medical Branch have leased one emergency hospital unit each. Prior to Operation Rebound, these units had been unpacked and repacked in functional form, each box or crate, for example, being painted on the corners with a distinctive color to indicate that its contents should go to triage, x-ray, laboratory, pharmacy, operating rooms, wards, supply, or utilities. Stored in huge vans, one of

This patient, like others evacuated from Houston, went through an advanced aid station. After preliminary treatment, he was brought to the hospital. Although Operation Rebound personnel recognized the seriousness of the trial, occasional gaiety relieved the long hours.

First stop at the hospital after admission was the triage station, where senior medical students under supervision of physicians examined the patient, noted the information on his tags, and ordered his next procedure. Caps of varied colors differentiated groups of participants.



Some patients, such as this one with severe facial lacerations, were examined by means of a portable x-ray unit. The unit used polaroid film which could be read two or three minutes after the picture was taken.

Rebound

which the Texas Medical Association has agreed to help finance, these hospital units will remain at Baylor and the Medical Branch but will be available for training purposes elsewhere in the state. Additional units are stockpiled at Camp Swift near Bastrop.

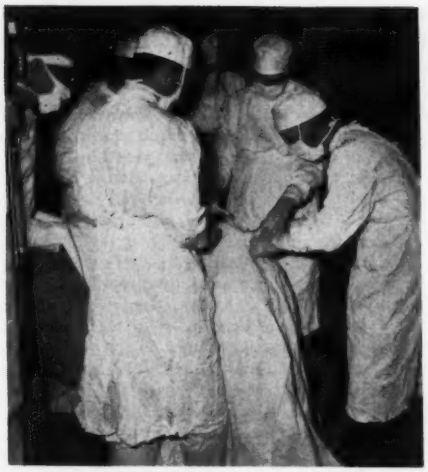
The hospital unit assigned to West Columbia was ready for patients 55 minutes after the van rolled to the door of the school building commandeered for the trial. This included a portable x-ray machine with polaroid film which gave finished prints two or three minutes after a patient was on the table. It also included an electric generator to furnish light for the hospital, sterilizers, sheets and blankets for the canvas cots, surgical masks and gowns, intravenous fluids, drugs, and a host of other equipment and supplies important to medical care in an acute emergency and for general care if necessary for a year or more.

In addition to the hospital units, aid stations, communications centers, and headquarters, a mobile blood bank was established in Alvin during the exercises, and a helicopter provided by the Army shuttled back and forth to carry supplies. Donors to the blood bank were the only visitors permitted to observe the trial other than official participants. Expendable supplies were conserved insofar as possible, only a minimum being actually used but others being brought into position and checked off so that an accurate determination might be made of what would have been used in circumstances other than imaginary.

A team of umpires from Brooke Army Medical Center, Fort Sam Houston, was on hand to analyze the effectiveness of Operation Rebound, and participants ranging from "patients" to physicians on the hospital staffs and official visitors were asked to fill out questionnaires on their roles and observations. The equipment, supplies, and care of patients will be evaluated and the data made generally available.

Major responsibility for staging Operation Rebound was assumed by Dr. Stanley W. Olson, dean, and Dr. J. R. Schofield, assistant dean of Baylor University College of Medicine, and Dr. Truman G. Blocker, Jr., professor of plastic and maxillofacial surgery at the University of Texas Medical Branch. Dr. Blocker served as commanding officer for the trial. Participating in the conference and acting as observers were about 50 members of Medical Education for National Defense, representing medical schools from throughout the United States; Dr. Schofield is national coordinator for this group.

Medical, surgical, and expectant wards were fully staffed with nurses to keep tab on the pulse and other symptoms important in continuing medical care. A laboratory and a pharmacy also were at hand.



In each of three operating units, patient, surgeon, and assistants carried out the motions of surgery.

Umpires from Brooke Army Medical Center and others evaluated each procedure to determine strong and weak spots in the hospital and its utilization. Here Col. George Prazak (standing left) confers with Dr. W. D. Nicholson, Freeport (white coveralls), who was chief of staff of the hospital unit at West Columbia; Dr. Ralph E. Gray, Lake Jackson (checked jacket); and other participants. Dr. C. F. Quinn, Texas City, was chief of the hospital at Arcadia.

Coordination of the entire operation was made possible by communications centers set up and manned by Civil Defense personnel.

Texas Medical Examiners Make Activities Report

Interstate licensure by reciprocity is now almost 100 per cent, a recent report of the Texas State Board of Medical Examiners indicates, with only Florida refusing to reciprocate with, or endorse, physicians from any of the other states. Texas reciprocates with, or endorses, all others and the District of Columbia.

Copies of a table showing the states and their reciprocal policies were recently sent by the Texas Board to the deans and the senior class presidents of the 85 medical schools in the United States. At the same time, photostatic copies of a front page *Houston Chronicle* editorial commending the Board for its holding of open meetings were sent to all the State Boards of Medical Examiners in the country.

The Department of Internal Revenue has rendered a decision making the Educational Council for Foreign Medical Graduates income tax free, the report also shows.

This council, composed of two representatives each from the American Medical Association, the American Hospital Association, the Association of American Medical Colleges, and the Federation of State Medical Boards of the United States, is set up to screen and examine foreign medical graduates from the 535 foreign medical schools, so that well-qualified foreign medical graduates may be licensed in this country.

Representative of the Federation of State Medical Boards on this council for the next four years is Dr. M. H. Crabb, Fort Worth, secretary of the Texas Board.

Burney Gives Tips to Lick Temporary Salk Shortage

In local areas where Salk vaccine shortages now exist due to the tremendous interest aroused by the recent doctors' campaign, three steps recommended by Surgeon General Leroy Burney of the Public Health Service may help make use of the existing supply most effective.

Preference should be given to those under 20 and to pregnant women. Secondly, local communities should begin their programs with first injections as supplies become available, without waiting to accumulate sufficient reserves to complete second and third injections. And, if planning groups would stagger dates of community vaccination drives, particularly in large cities, that also would help even out available supplies.

So far this year, more than 35,000,000 cc. have been used, compared with 77,000,000 in all 1956; production in January reached 11,000,000 cc., in February, 16,500,000, record highs. The surgeon general anticipated that the shortage would begin easing up by mid-April.

Governor Proclaims Special Observances

Future Nurse Day was proclaimed by Governor Price Daniel for March 30, the day on which the Future Nurses of Texas had their third annual meeting in San Antonio, and Health Education Week has been designated as April 28-May 4 by the Governor.

In calling attention to Future Nurse Day, the Governor said:

> Continued renewal and extension of the ranks of the nursing profession, through the attraction of young poeple into this vital work, is essential to the future health and welfare of the Nation.
>
> There are many problems facing the nursing profession which can be solved only through the aid of an informed and sympathetic public.
>
> . . . It is proper and fitting that on this day our attention and recognition should be focused on the great contributions, past and present, that the nursing profession has made to the national welfare and security.

Relating to Health Education Week, which overlaps the period of the Texas Medical Association annual session, the Governor's proclamation stated:

> The high standards of health and medical care enjoyed by the people of Texas have been achieved through the cooperation of the many branches of science, business, industry, education and all segments of society.
>
> The Health Fair, sponsored by the Woman's Auxiliary to the Texas Medical Association, April 28 through May 8 in Dallas, is a commendable effort to provide further health education for the people of Texas.
>
> Therefore, I, as Governor of Texas, do hereby designate the period of April 28-May 4, 1957, as Health Education Week in Texas, and urge all citizens to avail themselves of the knowledge offered by the Health Fair in Dallas.

Personals

Dr. Gilbert H. Fletcher, chief of the Department of Radiology, University of Texas, and director of radiation therapy, University of Texas M. D. Anderson Hospital and Tumor Institute, Houston, discussed "The Role of Radiation Therapy" at the tenth annual Connecticut Cancer Conference for Physicians, held March 13 in Hartford.

Among guest speakers at the ninth annual Mid-West Cancer Conference, held March 7-8 in Wichita, Kan., was *Dr. Vincent P. Collins* of Houston.

Dr. Frederick C. Rehfeldt of Fort Worth became president of the Congress of Neurological Surgeons at its recent annual meeting.

Joe F. Lakey, director of the Bureau of Food and Drugs, State Health Department, in Austin, has been appointed to an advisory committee on food standards, Commissioner George P. Larrick has announced from Washington, D. C. It is a division of the Food and Drug Administration.

Dr. Andrew S. Tomb, Victoria, chairman of the American Academy of General Practice Liaison Committee with the American Psychiatric Association, will participate in an hour-long, five-state, closed-circuit television seminar on "The Physician and Emotional Disturbance" May 6. The "Videclinic" will be presented by the American Medical Association's Council on Mental Health and the medical societies in the five states receiving the program, in cooperation with Smith, Kline and French Laboratories, Philadelphia.

Dr. John B. Trunnell, Houston, spoke on "Trends in Cancer Research" when he appeared at the third annual Seminar in Cancer presented by the medical staff of the Huron Road Hospital, Cleveland, in February.

Dr. W. Compere Basom, El Paso, was elected a member of the Board of Governors to the American Fracture Association in Chicago recently.

Dr. George C. Morris, Jr., of the Baylor University College of Medicine, Houston, has been selected as one of 25 Scholars in Medical Science by the John and Mary R. Markle Foundation. The sum of $750,000 has been appropriated toward support of the scholars in the schools where they will teach and carry on research; for each scholar ap-

pointed, the fund is $30,000, granted at $6,000 annually for five years. Surgery is the field of Dr. Morris, who attended Duke University and received his medical degree from the University of Pennsylvania School of Medicine.

Dr. A. B. Pumphrey, Fort Worth, was given a certificate of merit recently for having attended the New Orleans Graduate Medical Assembly 10 times, a distinction few physicians have achieved.

Dr. Samuel A. Shelburne, Dallas; *Dr. Paul E. Teschan,* Fort Sam Houston, San Antonio; and *Dr. Bryan Williams,* Dallas, were among featured speakers at the thirty-eighth annual session of the American College of Physicians, held April 8-12 in Boston.

Dr. F. J. L. Blasingame, Wharton, trustee of the American Medical Association, has been appointed one of two AMA delegates to the Istanbul meeting of the World Medical Association.

A resolution honoring the late *Dr. Sam E. Thompson,* Kerrville, was passed by the Texas House of Representatives in Austin January 24. It was introduced by Representative Joe Burkett, Jr., and signed by Speaker Waggoner Carr.

Dr. Jorge L. Ceballos, assistant professor of radiology at the University of Texas Medical Branch, is resigning from that position as of May 1 to return to his native country, Mexico. He and Mrs. Ceballos have lived in the United States for six years.

Dr. and Mrs. Wilber Forrest Robertson of San Antonio have announced the engagement of their daughter, Sarah Joe, to Philip Duder LeMessurier of Bloomfield Hills, Mich. An early fall wedding is planned by the couple, who are both seniors at Denison University, Granville, Ohio.

Dr. and Mrs. James W. Finney, San Antonio, recently became the parents of a son.

Dr. and Mrs. J. H. Playfair are parents of a son, Thomas Mitchell, born January 31 at Luling.

Hazards in Metal Arc Welding

The newer welding technique known as inert-gas-shielded consumable-electrode arc welding was first introduced about six years ago and has been coming into more general use in the past year. This method uses a shield of argon gas around the arc. A higher electric current density is required and a consumable electrode. These two factors make ultraviolet radiation 5 to 30 times as great, thus increasing the dangers of exposure to the skin and eyes and the spatial range of such hazards.

Ozone is produced by the ultraviolet radiation from the oxygen in the room. Ozone causes irritation of the eyes, nose, and throat; headache and lethargy results and in more serious concentrations, pulmonary complications. The oxides of nitrogen produced from the nitrogen in the air are increased when the ultraviolet radiation is increased, but less so than ozone. Metal fumes from the material being welded and the compound used in the welding are hazards. Aluminum oxide, nickel carbonyl, and oxides of chromium, lead, and zinc may be produced, depending upon the metal.

It is anticipated that this technique will increase because inert gas shield of helium or argon around the heat source avoids contamination of the weld by oxidation and reduces discoloration, splatter, and pitting. Continuous feed aspect of this welding process is also advantageous as it further reduces pitting and splatter, produces a smoother weld faster, and allows a longer seam to be welded without interruption.

Following are 3 cases reported as a result of the increased ozone concentration.

1. A 51 year old man suddenly developed headaches, substernal oppression and dyspnea while at work. On admission to the hospital, one hour later, he had frank pulmonary edema but no other evidence of heart failure. The electrocardiograms were normal. Chest roentgenogram showed diffuse peribronchial infiltration with peribronchial and multilobular pneumonia. Two weeks were required for his recovery under symptomatic management.

2. A male worker, age 47 years, noted burning of his eyes and throat for three days and developed a cough and a sensation of choking and dyspnea. Examination revealed bronchial breathing and coarse rales over both lung fields. The patient was hospitalized and treated for bronchial pneumonia.

3. A 31 year old male welder, after leaving work, noticed an acid taste and a severe sudden anorexia. That evening he had a shaky chill and his temperature rose to 102 F. He developed substernal oppression and dyspnea. He was treated with penicillin at home, and after nine days he was well. The early chest roentgenogram showed mottled densities in both lung fields.

All three of these workers had recently changed from the conventional arc-welding technique to the consumable electrode arc-welding machine. Control of such conditions is largely through good engineering practices with adequate ventilation and exhaust hoods close to the site of the welding. An awareness of the meaning of these symptoms would help point out the difficulty more quickly than was the case in the report.—Kleinfeld, M.; Giel, C.; and Tabershaw, I. R.: A.M.A. Arch. Indust. Health, 15:27-31 (Jan.) 1957.

Fresh Viewpoint

A new way of looking at the proposed federal budget for fiscal 1957 was suggested by columnist John McCutcheon in the Chicago *Tribune.* He translated the breakdown of the budget into terms of a family with a $10,000 income as follows:

> It hires a policeman for $6,200, mainly to keep an eye on No. 2 big shot, an unpredictable character who lives across town. It pays $1,050 interest on money borrowed to hire the policeman. It pays $750 to a retired policeman, and it gives $300 to neighbors who, it hopes, won't object when the policeman steps on their lawns, and may even help him at his job. With these items out of the way, there is $1,700 left for the use of the family.
>
> However, there are a number of sons who have been pretty expensive recently. One has really had a tough time, what with illnesses, etc., and he gets $450 a year to keep him going. The others are in one trade or another, such as farming, labor, and mining. In recent years, because of jealousies, parental meddling, etc., relations have become strained with their parents and with one another. They now get about $950 a year just to keep the domestic peace.
>
> The mother keeps the home going on what is left: $300.

From US to Korea: $76,000 in Medical Books

The American-Korean Foundation and the United States Army Medical Service have announced discontinuation of their joint project of shipping medical books contributed by American individual physicians and medical groups to Korea. Response to the appeal was so generous, states Dr. Howard A. Rusk, foundation president, that more than 77 tons of medical texts, references, and periodicals, valued at $76,000, have been shipped to Korea from the United States.

Arthritis

Arthritis is perhaps the oldest disease of which we have definite proof. Examples have been collected from fossils dating back to remote antiquity. Yet authorities say it is one of the most neglected diseases.

Baillou, in 1570, was the first to use the term "rheumatism" as a name for acute arthritis. He was also the first to regard rheumatism as a clinical entity. From 1672 comes a description of an arthritic condition from Richard Wiseman, surgeon to King Charles II. This was a description of a "tumor albus," which is now recognized as tuberculosis of the joints.

Thomas Sydenham in 1683 published his "Tractatus de podagra et hydrope," in which he clearly differentiated gout from rheumatism. Sydenham also distinguished between acute and chronic forms of arthritis.

Heberden was the first to describe the arthritic nodes, which are now called by his name.

Therapy under Hippocrates' methods was conservative. He suggested diet, baths, warm climate, purging, and local applications. Celsus advised hot fomentations to the joints, hot plasters, and bloodletting; for pain he prescribed poppy heads boiled in oil and mixed with wax salve made of rose oil. In respect to treatment, Sydenham was ahead of other physicians, as he recommended, "Early to bed and early to rise, keep the mind easy, exercise regularly, and refuse wine."

The book "Arthritis and Common Sense," published in 1956, which for several weeks was a best seller, has been labeled by the Federal Trade Commission in a complaint against the author and publisher, as containing "false, misleading, and deceptive statements." A complete review of this book may be found in the *Bulletin of the Los Angeles Medical Association* for January 17, 1957.

Rheumatic diseases are a common cause of chronic illness in this country and rank second in causing temporary and permanent disability. It is estimated that 1 out of every 20 persons in the United States has some form of rheumatic disease. Rheumatism is more common than tuberculosis, diabetes, cancer, or heart disease. It cripples without killing, and keeps more people away from work than accidents. More than 50 per cent of the disabled from this disease are people under 45 years of age.

The rapid progress being made in the treatment of rheumatic diseases is reflected in the following list of references available in the Texas Medical Association Memorial Library.

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Gifts to the Library

Dr. Luise C. Brandenstein, Kerrville, 74 journals.

Dr. C. H. Brown, Jr., Stephenville, 148 journals.

Dr. Morris Davidson, Austin, 5 books, 2 journals, 21 reprints.

Dr. J. Edward Johnson, Austin, 4 journals, 2 reprints.

Dr. Edgar F. Jones, Jr., and Dr. Marcus L. Ross, Galveston, 17 tape recordings.

Mrs. H. S. Leon, Austin, 6 journals.
Dr. William F. McLean, Austin, 29 books.
Dr. B. E. Pickett, Sr., Carrizo Springs, 4 journals.
Dr. Morris Polsky, Austin, 31 journals.
Dr. Nelson L. Schiller, Austin, 37 journals.
Dr. B. O. White, Austin, 39 journals.
Dr. David R. Womack, Austin, 95 journals, reprints.

New Tape Recordings

The following tape recordings were presented to the Library by Drs. Marcus L. Ross and Edgar F. Jones, Jr., of Galveston:

"Vaginal vs. Abdominal Hysterectomy"—Dr. John C. McDermott, Los Angeles.

"Anemias of Pregnancy"—Dr. Roy Holly, Omaha.

"The Elective Induction of Labor"—Dr. Edward H. Bishop, Philadelphia.

"Treatment of Carcinoma of the Cervix"—Panel discussion.

"Toxemias of Pregnancy"—Panel discussion.

"Surgical Problems of Pregnancy"—Dr. Ralph A. Reis, Chicago.

"Obstetric Hemorrhage"—Panel discussion.

"Prevention and Management of Prematurity"—Symposium.

"Antimicrobial and Chemotherapeutic Agents in Obstetrics and Gynecology"—Formal papers.

"Gynecologic Complaints of Immature Females and Teen-Age Obstetrics"—Dr. Goodrich C. Schauffler, Portland, Ore.

"Legal Aspects of Obstetric Practice"—Dr. Louis J. Regan, Los Angeles.

"Indications for Hysterectomy"—Panel discussion.

"Medical Complications of Pregnancy"—Symposium.

"Factors Determining Type of Cesarean Section"—Panel discussion.

"Management of Prolonged Labor"—Panel discussion.

"Indication for Surgical Treatment of the Ovary"—Dr. Clyde L. Randall, Buffalo.

"Lesions to Be Considered in the Differential Diagnosis of Carcinoma of the Cervix"—Panel discussion.

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★ Books

Treatment of Migraine

JOHN R. GRAHAM, M.D., *Chief, Medical Service, Faulkner Hospital; Clinical Associate in Medicine, Massachusetts General Hospital; Instructor in Medicine, Harvard Medical School, Boston. 149 pages. $4. Boston, Little, Brown and Company.*

A Hindoo fable prefaces this monograph and thereby neatly pictures medical attitudes on migraine. At the same time it creates a mellow mood for the reading of the comprehensive treatise of migraine that follows. After lamenting the fact that we still do not know what triggers an attack of migraine, the author spends some time on definition. He differentiates between what he calls "classic migraine," the headache associated with neurologic aura, and the more

common "atypical migraine" or sick headache. At the same time he warns against migraine as a quick diagnosis of any severe headache and details those that may more probably stem from some organic lesion. Case histories clarify his differential points.

Secure in the diagnosis of migraine, the author proceeds to therapy. He leans heavily on ergot and its derivatives, both for diagnosis and treatment. He explains the mechanism whereby ergot achieves its effect, takes note of its possible toxic effects, specifically lists the contraindications to its use, and then offers many helpful suggestions in the management of patients who show unpleasant side reactions to ergot. Again the use of case histories points up individual difficulties. Therapy other than ergot is listed but without enthusiasm.

The chapter on prevention emphasizes general hygiene, both physical and emotional, along with a sympathetic attempt at modification of the migrainous personality. The importance of careful history and personal interest is stressed. Finally, notice is taken of the manifold treatments offered by the specialties, allergy, psychiatry, endocrinology, and even surgery.

This book is well worth reading. The style is good and the content is valuable.

—Caroline Crowell, M.D., Austin.

Complications of Regional Anesthesia; Etiology, Signs and Symptoms, Treatment

DANIEL C. MOORE, M.D., *Director, Department of Anesthesiology, Mason Clinic, Chief of Anesthesia, Virginia Mason Hospital, Seattle. 291 pages. $10.50. Springfield, Ill., Charles C Thomas.*

For the past several years the author has been a leader and a real enthusiast in the field of regional anesthesia among American anesthesiologists. He is, therefore, thoroughly qualified to write with authority regarding its complications. It is unfortunate that more enthusiasts have not reported adequately their untoward results.

The author has pointed out in his introduction that his complications of regional nerve blocks are due primarily either to "untoward results of the normal pharmacological actions of the blocking solutions used, or to trauma associated with the technique." Upon this basis he has discussed the etiology, signs and symptoms, and treatment of each separate complication.

The book is divided into three parts. Part 1 considers the complications which are seen following local infiltration and peripheral nerve block; part 2 discusses in detail the complications associated with spinal and epidural block; and part 3 describes incidental complications such as broken needles, unsatisfactory anesthesia, and certain postoperative complications which may occur with any anesthetic technique.

The text is well illustrated with photographs which properly emphasize the extreme end results of some preventable complications. Certain information is presented in table form for rapid comparison of data and clarity of organization.

There is probably no other single work available which has so completely considered the complications of regional anesthesia as reported in medical literature from all over the world, both historic and current. This review of the literature plus the author's own outstanding experience has not overemphasized the dangers involved in the use of these techniques, but has clarified the low incidence of serious complications when respect is shown for the pharmacological properties of all drugs used and the possible trauma of the techniques involved.

This book should be read by every medical and dental student to give them proper respect for the possible complications of techniques too frequently taught as having little or no danger. Every practicing physician and dentist should read it to learn again the causes, symptoms, and proper treatment of the complications they may experience in their offices this very day.

—Ivey E. Lamberth, M.D., Tyler.

Handbook of Physical Therapy

ROBERT SHESTACK, PH.G.R.P., P.T.R., *Technical Director, Department of Physical Therapy, Washington County Hospital, Hagerstown, Md.; Director, Department of Physical Therapy, and Consulting Physical Therapist, Kings Daughters Hospital, Martinsburg, W. Va. Foreword by I. William Nachlas, M.D. 212 pages. New York, Springer Publishing Company, Inc., 1956.*

This book, written by a physical therapist, is an attempt to make a very specialized field simple and easily understandable. Unfortunately, the author has tried to cover, in this small volume, almost the entire gamut of physical agents as applied to a vast range of diseases, from laryngitis to peripheral vascular disease.

The book is apparently based on the author's clinical impressions, and many of his recommendations conflict with techniques generally accepted in physical therapy. For example, he recommends covering the skin when bakers or infrared lamps are used; he considers diathermy absolutely contraindicated in the treatment of stiff joints; he uses two forms of heat in the treatment of bursitis and tenosynovitis, after previously stating that one should not order several types of modalities at one time where one modality will suffice.

There is a section on treatment with galvanic current for conditions without denervation, although this technique is now obsolete. In the massage chapter, there is a section dealing with percussion movements, which are, of course, rarely if ever used in therapeutic massage. A number of exercises for different areas and joints are described with no indication for their use.

Similarly, there are questionable statements in the clinical conditions discussed. An example is the use of galvanic stimulation in the flaccid paraplegic. Such treatment can have no possible value in a patient with part of the spinal cord completely destroyed.

Since the recommendations of the author are questionable in so many instances, the reviewer believes that this book should be recommended only for the physician or physical therapist who has had sufficient experience to be able to abstract the worth-while portions of the book.

—Edward M. Krusen, M.D., Dallas.

Health for the American People

A Symposium, Presented Monday, November 21, 1955, Massachusetts Memorial Hospitals Centennial Celebration. Published for Massachusetts Memorial Hospitals, Boston. 105 pages. $1. Boston, Little, Brown and Company, 1956.

The Support of Medical Research

A Symposium Organized by the Council for International Organizations of Medical Sciences. Established Under the Joint Auspices of UNESCO and WHO. Edited by SIR HAROLD HIMSWORTH, *Chairman of the Conference, and* J. F. DELAFRESNAYE, *C.I.O.M.S. 170 pages. Springfield, Ill., Charles C Thomas, 1956.*

A History of the Therapy of Tuberculosis And the Case of Frederic Chopin

ESMOND R. LONG, M.D., *Director of the Henry Phipps Institute, University of Pennsylvania, and Director of Medical Research, National Tuberculosis Association. 71 pages. $2. Lawrence, University of Kansas Press, 1956.*

To the Clendenning Lecture Foundation we are indebted for the inspiration that gave us this clever and interesting story of the therapy of tuberculosis.

Speaking from his background in training as a pathologist, his experience as an investigator, and his career as a director of medical research in tuberculosis, Dr. Long is eminently qualified to tell this story. The reader used to the stilted and restricted vocabulary of the average medical writer is surprised and pleased at the literary resourcefulness shown by this author in his choice of words and in the clearness of his descriptions.

He uses the "Illness and Medical Care of Frederic Chopin" as an example of the helplessness of other times as contrasted with present progress. It is a clever ruse and works well in holding the interest of the reader while the low state of medical science of the times is unfolded in the vivid imagery of the sick room of the famous composer.

"There are three powerful influences in Chopin's short life," he tells us, "his music, his loves, and his tuberculosis." All three are brought into focus in this dramatic story.

Following this biography, an outline of therapy from Hippocrates to modern times is given. This is brief but complete for the purposes and use of the average medical reader.

The chronological outline of stages of progress with dates, names of investigators and pioneers, and a brief statement of their chief contributions is closed with a long list of references for further reading and a comprehensive index. It is truly a classic in medical history.

—J. Edward Johnson, M.D., Austin.

New Bases of Electrocardiography

DEMETRIO SODI-PALLARES, M.D., *Chief of the Department of Electrocardiography at the National Institute of Cardiology of Mexico; Professor of Cardiovascular Clinics at the National University of Mexico, Mexico City, with the collaboration of* ROYALL M. CALDER, M.D., *Ed., English translation, Clinical Professor of Medicine, Graduate School, Baylor University; Physician and Cardiologist, San Antonio. 727 pages. $18.50. St. Louis, C. V. Mosby Company, 1956.*

This is a book packed with the best information regarding the why and wherefore of an electrocardiogram. In no volume has this reviewer seen the amount of informative material so well presented in so few pages.

This is not a book on clinical electrocardiography, but is a compenium of scientific information upon which we can call for the interpretation of a clinical electrocardiogram.

It is well written and illustrated. The subject matter is arranged in chapters developing each phase in chronological order, so that the physical laws governing the electrical phenomena that take place in the myocardium are more easily appreciated. The cardiologist can reason out the changes in the tracing, determine the mechanism involved, and untangle the complexities that may be presented.

Sodi-Pallares, in conjunction with his staff, has done extensive research on many problems, such as ventricular overloading, ventricular activation, and bundle branch block. He was the first to obtain a tracing from the interior of the ventricular cavities in studying the activation of the myocardium. He also studied septal activation by means of double simultaneous catheterization, this being the first studies in man.

This book presents to the clinician an appendix full of simplified mathematical formulas arranged in sequence so that a busy cardiologist can save time by referring to them.

—Ben H. Cooley, M.D., El Paso.

The Office Assistant; In Medical or Dental Practice

PORTIA M. FREDERICK, *Instructor, Medical Office Assisting, Long Beach City College, and* CAROL TOWNER, *Executive Assistant, Department of Public Relations, American Medical Association. 351 pages. $4.75. Philadelphia, W. B. Saunders Company, 1956.*

As an excellent beginning to their book, the authors outline possibilities for women in the medical field, giving the qualifications, job possibilities, and duties. They cite intelligence, dependability, and personality as top qualifications for the ideal assistant. One chapter outlines the medical code of ethics and medicolegal principles, impressing the fact that the welfare of the patient is all important and comes before all other interests.

The subject of keeping adequate files and records is well covered as is the collection of fees and establishment of fair fees. These are certainly second in the physician's or dentist's practice to the care and treatment of patients. By having good office practices and topnotch bookkeeping, the doctor will encourage confidence by his patients.

Routines for physical examinations and office assisting techniques are well outlined, and from the material, the reader would be able readily to develop techniques that would fit into any practice. Not to be overlooked is the good discussion of sterilization techniques, for the patient is often more concerned about the cleanliness of instruments, tables, and office than the office assistant realizes. A messy office does little for the already low morale of a sick patient.

Drug terminology and preparation and administration of medicines are essential to the office assistant if she is to understand and carry out the doctor's orders. Medical terminology is also a must in any practice.

The discussion on library duties is well done, but in the ordinary office such work is neither desired nor required to the extent described. Of course, it would be well for the assistant to have a passing understanding of library methods, as she may be required to do some research from time to time in the medical library.

Persons in any profession will agree that a smoothly run office is the key to satisfaction on the part of the customer or patient and the employer or physician. The authors have given many valuable pointers on this subject. Many small and seemingly insignificant duties which in reality should not be overlooked are discussed in detail. Duties to make the office more pleasant and a place of which to be proud are expressed in such a manner as to be the peak of the entire book, and they cannot be overemphasized.

—Mrs. Mildred Foxworth, Medical Assistant, Austin.

Christopher's Minor Surgery

ALTON OCHSNER, M. D., F.A.C.S., *William Henderson Professor of Surgery and Chairman of the Department of Surgery, Tulane University of Louisiana School of Medicine, and* MICHAEL E. DEBAKEY, M. D., F.A.C.S., *Professor of Surgery and Chairman of the Department of Surgery, Baylor University College of Medicine. ed. 7, 547 pages. $9. Philadelphia, W. B. Saunders Company, 1955.*

Electrodiagnosis and Electromyography

SIDNEY LICHT, M. D., *Honorary Member, British Association of Physical Medicine, Danish Society of Physical Medicine, and French National Society of Physical Medicine, Ed. 272 pages. $10. New Haven, Elizabeth Licht, Publisher, 1956.*

This book contains 12 chapters written by 11 authors on the use of electricity in diagnosis and treatment of neuromuscular disorders. It places special emphasis on the basic physiologic principles of such procedures for clinical application. The first chapter, by Licht, describes the history of use of electricity in medicine; this is followed by a chapter on the various apparatus used. The third chapter, by Ernest Fischer, on the physiology of skeletal muscle, is one of the best discussions on this subject ever written. It should be read by all physicians who are treating neuromuscular or neurologic disorders. Other chapters are on motor points, routine electrodiagnosis, chronaxy, strength-duration curves, progressive and alternating currents, electromyography, and electrical skin resistance. In general, this book provides a good up-to-date detailed reference on the subject.

—Odon F. von Werssowetz, M. D., F.A.C.P., Gonzales.

Textbook of Medicine

RUSSELL L. CECIL, M. D., Sc. D., *Professor of Clinical Medicine Emeritus, Cornell University, New York; and* ROBERT F. LOEB, M. D., Sc. D., *Hon. Causa, LL.D., Bard Professor of Medicine, Columbia University, New York, Eds.;* ALEXANDER B. GUTMAN, M. D., Ph. D., *Professor of Medicine, Columbia University, New York;* WALSH MCDERMOTT, M. D., *Livingston Farrand Professor of Public Health and Preventive Medicine, Cornell University, New York; and* HAROLD G. WOLFF, M. D., *Professor of Medicine (Neurology), Cornell University, New York; Associate Eds. ed. 9. 1786 pages. $15. Philadelphia, W. B. Saunders Company, 1955.*

This comprehensive volume embodies the more important and fundamental facts of previous volumes and at the same time has reflected the many strides which have taken place over the past several years. The passing of former contributors has made necessary the transposition of some material and has included preparation of 58 new treatises on subjects previously covered. It is noteworthy that some 40 odd new diseases or conditions have been covered which were not previously included. Examples of some of the new subjects covered are Colorado tick fever by Florio; coxsackie viral infections by Huebner; cat scratch disease by Daniels; the leptospiroses by Beeson; milk sickness and the painful shoulder by Cecil; burning feet syndrome, pyridoxine deficiency, vitamins and blood regeneration, and kwashiorkor by Spies; atherosclerosis and xanthomatosis by Barr, and many others. The magnitude of this book is realized by the fact that it has two editors, three associate editors, and more than 170 contributors. The number of contributing universities came close to 50, with the greatest number of contributors coming from Harvard, Columbia, and Cornell Universities and the federal services, which includes the National Institutes of Health. One cannot help feeling that a few more prominent contributions from other lands might enhance its value.

The book is printed on excellent paper and is edited in an easily readable style. The reviewer heartily recommends it as a handy reference book for any practicing physician's book shelf.

—Joe C. Rude, M. D., Austin.

★ Texas Medical Association

Disability Insurance Plans Compared

A review of group disability insurance programs offered by 29 state medical societies, 5 large county societies, and the District of Columbia society has been completed by the Insurance Committee of the California Medical Association. Texas was one of the states.

The review showed that:

1. Maximum accidental death benefit offered in any contract is $5,000. The plan sponsored by the Texas Medical Association is one of 13 that offers $5,000.

2. The maximum age at which maximum benefits can be secured in any contract is 70 years. Texas has one of seven plans with a maximum enrollment age of 70.

3. The standard waiting period before disability benefits start is zero days for accidental disability and seven days for illness disability. Texas and California, which offer options from the first day to 180 days, have the widest range and the largest numbers of options.

4. Hospitalization insurance is offered in most of the contracts as an option at added cost. Texas has a maximum hospital benefit of 120 days, offered at added cost.

5. Texas is the lone state which offers benefits for illness up to 10 years and one of 12 which offers lifetime benefits for disability from accident.

6. Most contracts offer weekly benefits of $100. Texas provides the maximum of the 35 policies surveyed—$150 a week or $600 a month.

7. Texas and California offer the widest range of coverage. Texas offers a choice of three year or 10 year coverage with optional waiting periods ranging from first day to 180 day coverage.

8. Premiums vary considerably because of the variations in contract provisions. However, 22 plans provide for the same level premium payments by all members, whereas 13 programs have premiums based on age groups. Texas offers both level premium (10 year sickness policy) and step-rate premium (three year sickness policy).

Among the contracts reviewed, 13 underwriters were included. The Lumbermens Mutual Casualty Insurance Company, which underwrites the Texas Medical Association plan, underwrites three others out of the total of 35 contracts.

MEDICARE IN TEXAS

The House of Delegates of the Texas Medical Association will hear a progress report on the Medicare program in Texas at its meeting in Dallas during the annual session, April 27-May 1. Public Law 569 of the Eighty-Fourth Congress, which introduced the program, went into effect on December 7, 1956. All state medical societies are participating in Medicare, with two exceptions, Rhode Island, where the Army is paying doctors directly, and Ohio, where

the Army has contracted with an insurance company to handle the physicians' Medicare.

A report from Blue Shield, which was selected by the Association to serve as fiscal agent for the Medicare program, indicates that through March 28, Blue Shield had paid 3,756 claims for services rendered to beneficiaries of this program. As of this same date, $281,984.04 had been paid to Texas physicians by the fiscal agent. This is an average of $75.08 per case.

There has been a gradual increase in the number of claims received each week by Blue Shield and the percentage of Medicare cases handled in Texas is considerably more than all but one or two of the other states.

A progress report as of March 1 indicated that nationally Army dependents make up 25.5 per cent of the doctor claims, Navy 30.1 per cent, Air Force 41.7 per cent, and Public Health Service 2.7 per cent.

The Office of Dependent Medical Care is asking Texas, like other states participating in the act, to extend its contract from July 1, 1957, until it has been renegotiated next year. Texas negotiations are set for January.

★ County Societies

Liberty-Chambers—President, Dr. William L. Barnett, Cleveland; vice-president, Dr. Boyce E. Gibson, Hull; secretary-treasurer, Dr. Richard O. Clements, Liberty; delegate, Dr. A. R. Shearer, Mont Belvieu.

Lubbock-Crosby—President, Dr. Harold Warshaw; vice-president, Dr. Robert H. McCarty; secretary, Dr. A. W. Bronwell; treasurer, Dr. Clyde F. Elkins, Jr.; delegates, Dr. A. Lee Hewitt and Dr. Allen T. Stewart, Jr., all of Lubbock.

McLennan—President, Dr. Howard O. Dudgeon; vice-president, Dr. R. H. Baskin; secretary-treasurer, Dr. Milton Spark; delegate, Dr. T. M. Oliver, all of Waco.

Montgomery—President, Dr. E. E. Lenon; vice-president, Dr. Irving M. Watson, Jr.; secretary-treasurer, Dr. M. G. Hutchins; delegate, Dr. D. L. Sadler, all of Conroe.

Nacogdoches—President, Dr. Eugene S. Rogers; vice-president, Dr. James I. Allen; secretary-treasurer, Dr. Charles W. Coussons; delegate, Dr. Stephen B. Tucker, all of Nacogdoches.

Nueces—President, Dr. J. L. Barnard; vice-president, Dr. A. L. Lane; president-elect, Dr. H. E. Griffin; secretary, Dr. John Lingenfelder; treasurer, Dr. Haddon B. Woods, all of Corpus Christi.

Orange—President, Dr. Howard C. Williams; vice-president, Dr. Homer C. Stuntz; secretary-treasurer, Dr. D. H. Pollock; delegate, Dr. F. W. Wilson, all of Orange.

Panola—President, Dr. Fred T. Hargrove; vice-president, Dr. Glen R. Johnson; secretary-treasurer, Dr. K. C. Prince; delegate, Dr. W. C. Smith, all of Carthage.

Pecos-Jeff Davis-Presidio-Brewster—President, Dr. John W. Pate, Alpine; vice-president, Dr. William E. Lockhart, Jr., Alpine; secretary, Dr. John C. Hundley, Fort Stockton; treasurer, Dr. George A. Hoffman, Fort Stockton; delegate, Dr. D. J. Sibley, Jr., Fort Stockton.

Polk-San Jacinto—President, Dr. Joseph Dabney, Jr., Livingston; president-elect, Dr. P. C. A. Singleton, Corrigan; vice-president, Dr. Robert L. Kurth, Livingston; secretary-treasurer, Dr. T. L. Gardner, Livingston; delegate, Dr. J. H. Dameron, Livingston.

Randall-Deaf Smith-Parmer-Castro-Oldham-Swisher—President, Dr. L. B. Barnett, Hereford; vice-president, Dr. W. B. Childress, Tulia; secretary-treasurer, Dr. W. D. Lawrence, Hereford.

Red River—President, Dr. James L. Wright, Clarksville; vice-president, Dr. Melvin Mark, Jr., Clarksville; secretary and delegate, Dr. E. E. Brooks, Bogata.

Taylor-Jones—President, Dr. C. A. McFadden; vice-president, Dr. W. Hubert Seale; secretary-treasurer, Dr. Tom C. Burditt; delegate, Dr. R. W. Varner, all of Abilene.

Tom Green-Eight—President, Dr. Sam H. Gainer; president-elect, Dr. K. B. Round; vice-president, Dr. Lloyd R. Hershberger; secretary-treasurer, Dr. Francis M. Spencer; delegate, Dr. R. G. Boster, all of San Angelo.

Top-O-Texas—President, Dr. R. M. Brown, Pampa; president-elect, Dr. W. G. Stevens, Borger; secretary-treasurer, Dr. Joe F. Brothers, Borger; delegate, Dr. Joe R. Donaldson, Pampa.

Washington-Burleson—President, Dr. Robert A. Hasskarl, Brenham; vice-president, Dr. W. A. Knolle, Brenham; secretary-treasurer, Dr. C. E. Southern, Brenham; delegate, Dr. G. V. Pazdral, Somerville.

Webb-Zapata-Jim Hogg—President, Dr. J. S. Reitman; vice-president, Dr. William J. Emerson; secretary-treasurer, Dr. L. G. Cigarroa; delegate, Dr. J. G. Cigarroa, Jr., all of Laredo.

Wharton-Jackson-Matagorda-Fort Bend—President, Dr. Lorraine I. Stengl, El Campo; vice-president, Dr. Stanley E. Thompson, Richmond; secretary-treasurer, Dr. Joseph N. Bader, Edna; delegate, Dr. L. B. Johnson, El Campo.

Doctors and Lawyers Meet Jointly

Members of the bar were guests of Pecos-Jeff Davis-Presidio-Brewster Counties Medical Society at a dinner meeting March 5 in Fort Stockton. The program consisted of a talk by an attorney, W. O. Shaeffer of Odessa, on "The Medical Witness" followed by a motion picture on this same subject.

A joint committee of members from the Travis County Medical Society and the Travis County Bar Association presented a program on "Medical Testimony in Damage Suits" March 19 in Austin. Using as a model the American Medical Association's motion picture, "The Medical Witness," but modifying it to suit the locale, the attorneys and physicians constructed a hypothetical damage suit in which an orthopedist was cross-examined as an expert witness. The testimony was given twice, once with the physician making the most common errors occurring in such testimony and the second time with him following the procedures dictated by good medical and legal standards.

Tarrant Doctors Endorse Medical Examiner System

A resolution endorsing the medical examiner system as an improvement over the lay coroner system now functioning in Texas was adopted by the Tarrant County Medical Society recently. The resolution called attention to the fact that investigation of death without medical attendance is primarily a medical problem and that use of competent physician examiners elsewhere in the nation has brought a high quality of investigations. Support of certain medical groups currently seeking to supplant the coroner system by the medical examiner system was urged.

Bosque-Hamilton Counties Societies to Combine

The Bosque County Medical Society, meeting March 14 in Clifton, voted unanimously to accept the request of the two residual members of the former Hamilton County Medical Society to consolidate.

Cameron-Willacy Physicians Consider Medicare

Dr. Roy Lester, Dallas, medical director of Group Hospital Service, spoke on the "Practical Considerations of the Medicare Program of Importance to Practicing Physicians," and C. Lincoln Williston, Austin, executive secretary of the Texas Medical Asosciation, discussed the "Dependent Medicare Program and Its Long Range Implications," when the Cameron-Willacy Counties Medical Society met February 18 in Harlingen.

Harris County Doctors Sponsor Scouts

Seventeen Harris County physicians served as sponsors for Eagle Scouts who have indicated an intention of studying medicine at an Eagle Recognition Dinner held March 8 in Houston. In addition to the time and money already devoted to this cause by the doctors, they will continue to counsel the Scouts.

WOMAN'S AUXILIARY

TEXAS' FIRST HEALTH FAIR

Texas' first Health Fair will be held at the Dallas Health Museum on the State Fair grounds April 28-May 5 in conjunction with the Texas Medical Association meeting. Twenty-two special exhibits are being prepared on the most pertinent and timely problems in the various fields of medicine. The 40 permanent exhibits of the Health Museum also will be on display.

For those who have not visited the Dallas Health Museum, the third of its kind in the United States and containing one of the two transparent men in existence, the coming Health Fair will be doubly rewarding. It should be noted that Texas' first Health Fair is the second state health fair ever staged, and the first offered to the public without charge.

The Health Fair is to help the individual develop an increased awareness about his health, to help him develop a common-sense attitude toward the problems of ill health, and to encourage him to seek competent advice when needed.

Even though the fair is staged for the general public, it is designed as well to aid physicians and their wives in serving their own community. What is a better way to offer some sound health education fact than an exhibit at the local county fair?

To avoid the stilted feeling often associated with scientific exhibits, the museum will be decorated in a fair motif. There will be free drinks and door prizes. The newest unit of the museum, the planetarium, will have special showings daily for the Health Fair visitors. There will be a special free show for the Woman's Auxiliary on Sunday, May 28.

Representatives of the Dallas County Medical Society have been consulted as to the choice and nature of the exhibits. Exhibits will be shown by the University of Texas medical schools in Galveston and Dallas, Baylor University College of Dentistry, American Medical Association, Dallas Veterinary Medical Association, Hensley Air Force Base, Texas State Department of Health, Dallas Health Department, Texas Division of the American Cancer Society, Dallas County Red Cross, Dallas Dietetic Association, Abbott Laboratories and the American Foundation for Allergic Diseases, Dallas Mental Health Society, Wadley Blood Center, Dallas Heart Association, Dallas Crippled Children Hospital, Children's Development Centers, Dallas Civil Defense, the Future Nurse Clubs of Dallas County, and winners of the District V Science Fair. On the patio will be the Dallas tuberculosis mobile truck and a civil defense disaster truck.

The Health Fair Committee, composed of Mrs. Jackson H. Speegle, chairman, Mrs. Albert H. Keene, Mrs. Thomas S. Barnes, and Mrs. Milford O. Rouse, all of Dallas, extends to all an invitation to "Come to the fair."

—MRS. JACKSON H. SPEEGLE, Dallas.

Officers of the Woman's Auxiliary to the Texas Medical Association for 1956-1957: President, Mrs. Richard C. Bellamy, Liberty; President-Elect, Mrs. H. S. Renshaw, Fort Worth; First Vice-President, Mrs. Harold Lindley, Pecos; Second Vice-President, Mrs. Ramsay H. Moore, Dallas; Third Vice-President, Mrs. William D. Nicholson, Freeport; Fourth Vice-President, Mrs. Lynn Hilburn, Henderson; Fifth Vice-President, Mrs. R. B. G. Cowper, Big Spring; Treasurer, Mrs. William C. Barksdale, Borger; Recording Secretary, Mrs. J. L. Jinkins, Galveston; Corresponding Secretary, Mrs. E. R. Richter, Dayton; Publicity Secretary, Mrs. Joe Thorne Gilbert, Austin; Parliamentarian, Mrs. John C. Parsons, San Antonio; Executive Secretary, Miss Hazel Casler, Austin.

One of the exhibits—on the eye—to be displayed at the Health Fair.

Exhibit to Illustrate Projects

The Woman's Auxiliary to the Texas Medical Association, at the request of the Association's Committee on Public Relations, will have a display at the annual session in Dallas this month to dramatize the many worth-while projects conducted by Auxiliary members.

Designed and constructed by members of the Cameron-Willacy Auxiliary, wire figurines in proper attire will illustrate the work done in behalf of public relations, legislation, nurse recruitment, organization, *Today's Health*, philanthropic funds, science fairs, Library, and AMA hostess.

Located on the mezzanine of the Baker Hotel, the exhibit will be staffed with well-informed Auxiliary members who will greet visitors and answer inquiries.

An extra feature of the Auxiliary exhibit will be the specially designed Christmas card, to be sold to doctors and their families, to aid the American Medical Education Foundation.

This year's card will be shown for the first time, and orders for the cards will be placed in a model of a medical school. The lights in the school will go on each time an order is placed inside and it is the hope of the Woman's Auxiliary that a flow of orders will keep the lights forever burning in free medical schools in America.

County auxiliaries will receive credit toward the annual AMEF award for purchases made by their members. The cards will be available in packets of 25 for $2.50.

DR. ROLAND EDWARD BING, SR.

Dr. Roland Edward Bing, Sr., of Oakwood, Texas, died at his home on December 22, 1956, of a heart attack.

He was born on August 27, 1886, in Sealy, Texas, and was the son of the late Henry and Ione Milton (Sledge) Bing. Two years later, the family moved to Waller County, where he received his early education.

Dr. Bing obtained his medical degree from the University of Tennessee in Nashville in 1909 and did postgraduate work at Tulane University in New Orleans. He began his practice in Bastrop, Texas, but after a few months was petitioned to come back to Waller County, where he then practiced for 13 years. In 1922, he moved to Oakwood, where he practiced until his death.

Dr. Bing was a member of the Texas and American Medical Associations throughout his career through the Bastrop, Waller, and Anderson-Houston-Leon Counties Medical Societies. He was county health officer; local physician and surgeon for the Missouri Pacific Railway; an examining physician for Selective Service in World Wars I and II, for which he received certificates of appreciation from the late President Franklin D. Roosevelt; and a member of the Presbyterian Church.

The physician and Miss Evalyn Winfree were married December 25, 1917.

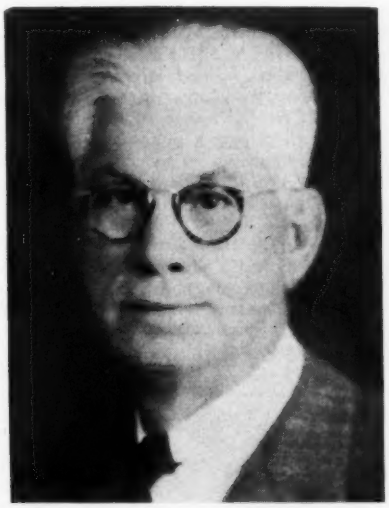
Survivors are Mrs. Bing; one daughter, Mrs. Robert S. Allen of Wichita, Kan.; three sons, Dr. Roland E. Bing, Jr., of Victoria, Dr. Lyndon W. Bing of Katy, and Dr. Arlys W. Bing of Fairbanks; a brother, L. B. Bing of Pasadena; and four grandchildren.

An obituary ordinarily will not be published more than four months after date of death. Cooperation in reporting deaths of physicians and in furnishing appropriate biographical material promptly is solicited.

DR. WILL P. ROGERS

Dr. William Peyton Rogers, El Paso, Texas, died January 20, 1957, in his home, of carcinoma of the right lung.

The son of Dr. H. E. and Sarah Emma Rogers, Dr. Rogers was born in 1875 in Lebanon, Tenn., and was graduated from Vanderbilt University School of Medicine, Nashville, in 1899. After beginning his medical practice in Milford, he moved in 1913 to El Paso, where he remained throughout his career. For a brief time, he was associated with the Mexican Northwestern Railroad and Affiliated Company Hospital in Madera, Chihuahua. He had been federal jail physician in El Paso continuously from 1916 until 1954. On his retirement from that position, he received high praise for his efficiency and consideration in caring for thousands of prisoners.



DR. WILL P. ROGERS

He had been a member of the Texas Medical Association since 1906 through the Ellis or El Paso Counties Medical Societies, and was elected to honorary membership in the state group in 1954. He was president of El Paso County Medical Society in 1937, and headed the professional division of the 1946 fund drive for the Hotel Dieu building project. He was on the staffs of Hotel Dieu, Providence, Old Masonic, and City-County Hospitals. A Scottish Rite Mason, he was presented a 50-year pin from the state Grand Lodge in 1956.

He was a captain in the Army Medical Corps during World War I, and served on the Procurement and Assignment Board for the Army and Navy during World War II.

Survivors include Mrs. Rogers, the former Miss Lula B. Adans, whom he married December 26, 1923, in El Paso; a son, William Hugh Rogers; a daughter, Mrs. Paul Harvey, Jr.; and three grandchildren, all of El Paso.

DR. AUSTIN OLIVER HULL

Dr. Austin Oliver Hull, who for 16 years was city bacteriologist of San Antonio, Texas, died of thrombosis in that city on January 28, 1957.

Dr. Hull was born on September 7, 1891, in Grand Cain, La. He was the son of John Colbert and Lillie Hull.

He attended Nacogdoches elementary schools, graduated from Nacogdoches High School, attended Tulane University in New Orleans, and received his medical degree from the University of Louisville in 1913.

Dr. Hull was a member of the Bexar County Medical Society, Texas Medical Association, and American Medical Association. He had practiced medicine in Three Rivers in 1914, and since that time in San Antonio, although he had been inactive the past few years.

He was married to Mrs. Esther Allsup Ralls in San Antonio in 1934.

Surviving Dr. Hull are his wife; one stepdaughter, Mrs. Floyd Price; a brother, Thomas P. Hull; and three grandsons, Jack Austin, Steven Craig, and Robert Bradley Price, all of San Antonio.

DR. J. H. HAMILTON

Dr. James Harvie Hamilton, Gatesville, Texas, died in McClosky Hospital December 5, 1956, of coronary thrombosis.

Son of James H. and Mary Elizabeth (McDill) Hamilton, he was born December 16, 1884, in Camargo, Tenn. He received his preliminary education in the public schools of Lincoln County, Tenn., and attended Morgan Prep School, Fayetteville, Tenn. He was a professional baseball pitcher for five years and school teacher for three years before turning to the field of medicine. He was graduated from Vanderbilt School of Medicine, Nashville, Tenn., in 1916. After serving two years as a first lieutenant in the Medical Corps Air Service, he did general practice in Jonesboro, Texas, from 1918 to 1926. In 1927 he moved to Gatesville, where he practiced until his retirement in 1952.

Dr. Hamilton had served several terms as president of the Coryell County Medical Society and was a member of the Texas Medical Association (honorary since 1953), American Medical Association, Masonic Lodge, and Odd Fellows Lodge. He was a school board member at Jonesboro. After moving to Gatesville he served six years on the city council, was on the board of the State Juvenile Training School for Boys, and was a trustee for Gatesville School District. He was also chief of staff at Coryell Memorial Hospital and a member of the board of stewards of the First Methodist Church.

Dr. Hamilton's other activities included farming, hunting, fishing, and playing golf. He was an inveterate sports fan and loved football and baseball. His hobby was developing the farm land which he had acquired.

The former Miss Mary Lucile Woodard and Dr. Hamilton were married in Nashville, Tenn., on July 16, 1915. She survives, as do three daughters, Mrs. Floyd Zeigler of Gatesville, Mrs. Ruth Cummings of Dallas, and Mrs. Sam Franks of Fort Worth; one sister, Mrs. D. A. Esterquist of Denver; and seven grandchildren.

DR. J. H. HAMILTON

DR. HENRY R. MARESH

Dr. Henry Rudolph Maresh, Houston, Texas, died in a local hospital January 28, 1957, after an illness of three months.

Dr. Maresh, son of Frank and Mary (Jerabek) Maresh, was born October 9, 1890, in Caldwell, and attended public schools in Weatherford. He attended the University of Texas, Austin, and was graduated from the University of Texas Medical Branch, Galveston, in 1919. He interned at St. Mary's Hospital, Galveston, and was a resident in internal medicine at Christ Hospital, Jersey City, N. J., and St. Joseph's Hospital, Houston.

DR. HENRY R. MARESH

In 1921, he began medical practice in Houston with his brother, Dr. R. E. Maresh, and continued this association throughout his medical career. He was a member of the Texas and American Medical Associations through the Harris County Medical Society, and was a member of Alpha Omega Alpha honorary medical fraternity, Texas and Harris County Heart Associations, Texas and Harris County Historical Associations, Doctors Club, Houston Club, Pine Forest County Club, and Riverside Methodist Church, of which

he was past president of the board of stewards and past chairman of the board of education. He was a member of the staffs of St. Joseph's and Methodist Hospitals, and was on the visiting staff of Hermann Hospital. He was a consultant in internal medicine at Polly Ryan Memorial Hospital, Richmond, and was assistant professor of clinical medicine at Baylor University College of Medicine. Dr. Maresh was the founder of the Edna Gladney Foundation, a charitable institution, and was a leader in affairs of Czechs, having been president of the Czech Educational Foundation of Texas and the author of "The Czech Pioneers of the Southwest." He contributed to the "Houston Book" and the "Texas Handbook," and helped edit the "Encyclopedia Slavonica."

Dr. Maresh also was active in the business field; he was president of the Liberty Loan and Building Association and owned the Maresh Machine and Welding Company.

He served in the Army Medical Corps during World War I, and was a Selective Service examiner during World War II.

Miss Thelma Burnett and Dr. Maresh were married September 2, 1933, in Dallas. Mrs. Maresh, Houston, survives. Other survivors include four children, Karele Norean, Haden Rene, Harlan Renan, and Marsha Burnett, all of Houston; two brothers, Dr. R. E. Maresh, Houston, and Frank Maresh, Rosenberg; and a sister, Mrs. R. A. Newman, Fort Worth.

DR. B. F. STOUT

Dr. Beecher Franklin Stout, a pioneer pathologist of San Antonio, died January 24, 1957, of cardiovascular failure after a prolonged illness. Dr. Stout was born in Baldwin City, Kan., May 27, 1877, the son of the Rev. Andrew V. and Anne (Dean) Stout. He was reared in Olathe, Kan., and there received his preliminary education. After being graduated from the University of Kansas School of Medicine, Kansas City, in 1900, he served his internship at St. Margaret's Hospital, Kansas City, and returned to the university for postgraduate training in pathology.

In 1904, Dr. Stout came to San Antonio where he established the first private laboratory for clinical pathology in Texas. Shortly thereafter he was credited with the first laboratory diagnosis of hookworm infestation in Bexar County. In 1907 he was appointed city bacteriologist and in 1909 served a term as physician to the San Antonio Public Schools. He was for many years a member of the San Antonio Board of Health and a member of the board of directors of the Robert B. Green Memorial Hospital. In 1910, Dr. Stout took postgraduate work at the Charite Hospital, Berlin, Germany, where he learned the then new technique of the Wassermann complement fixation test for syphilis. Returning to San Antonio in 1911, he performed the first Wassermann test in Texas and administered the first dose of salvarsan. Later he took additional postgraduate training in pathology at Cornell University Medical College, New York.

DR. B. F. STOUT

During his 50 years of practice, he served as pathologist to the Robert B. Green Memorial Hospital, the Nix Memorial Hospital, and the Salvation Army Hospital and as consulting pathologist to the old Physicians and Surgeons Hospital and to other hospitals over Southwest Texas.

Dr. Stout had been a member of the Texas and American Medical Associations through Bexar County Medical Society since 1904. He served as secretary to his county society in 1904 and was elected honorary president in 1920. In 1933 and 1943 he served as chairman of the Section on Clinical Pathology of the Texas Medical Association. He was elected to honorary membership in the state Association in 1955. He was responsible for instituting the first tumor seminar in San Antonio in 1944. It was so successful that it was repeated annually and has evolved into one of the nation's outstanding tumor conferences, now sponsored by the San Antonio Society of Pathologists. Dr. Stout was a diplomate of the American Board of Pathology, a fellow of the American Society of Clinical Pathologists, and a founder and charter member of the Texas Society of Pathologists.

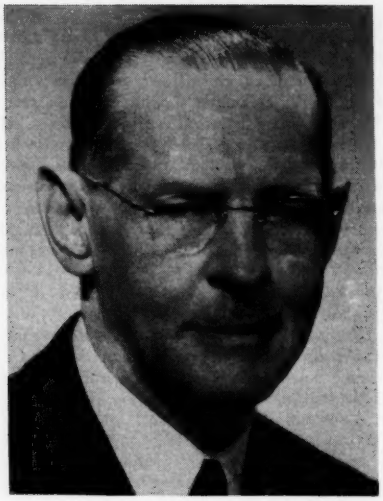
Unable to join the Army during World War I because of a serious hearing defect, Dr. Stout became contract clinical pathologist to the Medical Department of the United States Army, serving as chief of the Serologic Service at Station Hospital, Fort Sam Houston. Here his work was so outstanding that Dr. John A. Kolmer invited Dr. Stout to write the chapter on serodiagnosis of syphilis for his textbook of clinical pathology. In January, 1956, Dr. Stout was selected by the Texas Society of Pathologists to receive the George Caldwell Memorial Award for outstanding contribution to the advancement of medicine in Texas. Dr. Stout was the author of numerous scientific medical papers.

Dr. Stout was married to Miss Llora Beach of Kansas City in 1905. He is survived by his wife; one daughter, Mrs. Frank R. Newton, Jr.; two granddaughters, Anne and Cynthia Newton; and a niece, Mrs. Martin C. Giesecke, all of San Antonio.

DR. GUY F. WITT

Dr. Guy Ferguson Witt, Dallas, Texas, died February 14, 1957, in a local hospital.

He was born in Eddy, Texas, January 24, 1883, and was the son of Dr. J. M. and Mattie (Ferguson) Witt. His preliminary education was in public schools in Salado and Bartlett, and he also attended the University of Texas, Austin, from which he was graduated in 1904. He received his medical degree from the University of Texas Medical Branch, Galveston, in 1911, and interned at the John Sealy Hospital there. His four years of residency in neuropsychiatry were spent at the San Antonio State Hospital; he also took postgraduate work in neuropsychiatry at Harvard University Medical School, Boston, in 1936. Dr. Witt did general practice in Waco from 1912 until 1915, and in 1919 he began his practice of neuropsychiatry in Dallas, where he remained throughout the rest of his career. With



DR. GUY F. WITT

Dr. James J. Terrill, he founded the Timberlawn Sanitarium, and was its medical director until the time of his death. He had been head of the Department of Neurology and Psychiatry at Baylor University College of Medicine for 20 years when that school was in Dallas and was professor emeritus and former head of the Department of Neuropsychiatry at Southwestern Medical School, Dallas. He also was chief of psychiatric services for Parkland and Baylor Hospitals until about two years ago. He was a staff member of the Methodist Hospital, and held membership in the Texas Medical Association, Southern Medical Association, American Medical Association, American Psychiatric Association, and Central Psychiatric Association and the Presbyterian Church. He was a past president of the Dallas County Medical Society, and was a charter member and past president both of the Texas Neuropsychiatric Association and the Southern Psychiatric Association.

During World War I, he served with the United States Navy as a lieutenant and was in charge of the neuropsychiatric section of a naval base hospital in France.

The former Miss Stella E. Lacy and Dr. Witt were married in Waco June 24, 1915. Survivors include Mrs. Witt; a son, Guy F. Witt, Jr., Dallas; a daughter, Mrs. Richard A. Slack, St. Louis; three brothers, Edgar E. Witt, former lieutenant-governor of Texas and now of Washington, D. C.; J. Leslie Witt, Houston; and Bertrand D. Witt, Austin; and one sister, Mrs. Mabel Witt Mansfield, Austin.

DR. M. C. HAGLER

Dr. Menan C. Hagler, New Braunfels, Texas, died in a local hospital January 6, 1957, of coronary thrombosis after a prolonged illness. He had retired from active medical practice in 1952.

He was born January 7, 1876, in Purdy, Mo. His parents were John A. and Dolly (Fly) Hagler. After attending public schools, Dr. Hagler became a school teacher at the age of 16, then worked toward being a railroad engineer. Later, he decided to become a doctor, and was graduated from the Kentucky School of Medicine, Louisville, in 1905. He began his practice in Monett, Mo., but moved in 1913 to New Braunfels, where he remained in active practice until the time of his retirement.

Dr. Hagler was a member of the Comal County Medical Society, and had served as its president in 1931, 1940, and 1941. He was a member of the Texas Medical Association and American Medical Association, and had served as city and county health officer and as railroad physician for local lines. He was a past president of the New Braunfels Chamber of Commerce and Lions Club, and had been a member of the New Braunfels school board, American Legion, Woodmen of the World, Elks Club, Jesters, and First Methodist Church. He was a thirty-second degree Mason and a member of the Shrine and Eastern Star.

DR. M. C. HAGLER

During World War I, he served as a captain in the Army Medical Corps.

Miss Ella McGuffin and Dr. Hagler were married November 28, 1906, in Monett, and Mrs. Hagler died in March, 1942. Although they had no children of their own, Dr. and Mrs. Hagler took a niece and a nephew into their home at different times when the death of a parent had occurred.

Survivors include a brother, L. E. Hagler, Bourbon, Mo., and several nieces and nephews.

DR. F. E. McALISTER

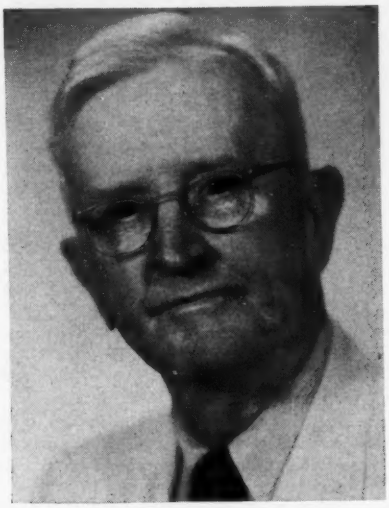
Dr. Finis Ewing McAlister, who had practiced medicine in Houston, Texas, until very recently, died January 6, 1957, in Mobile, Ala.

Dr. McAlister was born in Woodville November 19, 1878, and was the son of Samuel and Sophronia (Young) McAlister. After attending preliminary schools, he entered the University of the South, Sewanee, Tenn., and was graduated in 1905. Four years later, he received his doctor of medicine degree from the University of Nashville, Tenn., and interned at Nashville General Hospital and at Tulane University Hospital, New Orleans. Later, he took postgraduate work at Tulane, Columbia University School of Medicine, New York, and Vanderbilt University School of Medicine, Nashville. Dr. McAlister practiced briefly in Victoria, Moscow, Bering, and Texla until 1917, when he

moved to Wiergate. He remained there until 1944, when he became associated with the Houston Clinic and continued to practice there until his recent illness. He had served as chief surgeon for the Gulf and Northern Railroad and for the Wier Long Leaf Lumber Company, Wiergate, for 25 years.

A member of the Texas and American Medical Associations through the Jefferson or Harris Counties Medical Societies since 1918, he also was a member of the Southern Medical Association.

Dr. McAlister was married to Miss Ida Elizabeth Slevin in Shreveport, April 10, 1908. She survives as do a son, Robert Ewing McAlister, Mobile, Ala.; two daughters, Mrs.



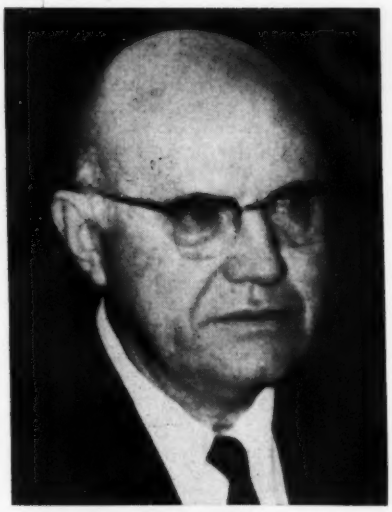
DR. F. E. McALISTER

Binford Sykes, Wilmette, Ill., and Mrs. Brack Hattler, Panama City, Panama; four brothers, Booth, John, and Samuel McAlister, Woodville; and Ralph McAlister, Nacogdoches; four sisters, Miss Altie McAlister, Woodville; Mrs. James O'Daniel and Mrs. Hugh Perry, Fort Worth; and Mrs. Floyd Smith, Austin; three grandsons, two of whom are preparing for medical careers; and four granddaughters.

DR. J. W. FOY

Dr. James Woodward Foy, Seymour, Texas, died January 10, 1957, in a local hospital after suffering a stroke some months before.

He was born July 12, 1901, in Anson; his parents were Alfred Lewis and Maggie (Easterling) Foy. After moving with his parents to Rotan when he was a small child, he attended public schools there. He also attended Tulane University, New Orleans, before entering Valparaiso University, Valparaiso, Ind. Dr. Foy was graduated from Baylor University College of Medicine, Dallas, in 1926; interned at Providence Hospital, Waco; and took his residency training at the Deaconess Hospital, Cincinnati. He practiced briefly in Quitaque and Rotan before moving in 1931 to Seymour, where he was in active practice until the time of his illness. He was largely responsible for the establishment of the Seymour Clinic and for an addition which was made to the Baylor County Hospital.



DR. J. W. FOY

Dr. Foy had been a member of the Texas Medical Association almost continuously since 1929 through the Baylor-Knox-Haskell Counties Medical Society, which he had served as secretary four years and president one year. He was a past president of the Thirteenth District Medical Society and the Seymour Chamber of Commerce, and was on the board of directors of the latter organization for several years. He also was a member of the American Medical Association, Chi Zeta Chi medical fraternity, and the Methodist Church.

Dr. Foy and Miss Lela Overstreet were married October 19, 1928, in Bowie. They had one son, James Woodward Foy, Jr., who is a premedical student at Texas Christian University, Fort Worth. Survivors other than his wife and son are his father, Alfred Lewis Foy, Rotan; a brother, Lewis H. Foy, Rotan; and three sisters, Mrs. Mary Grindstaff, Fort Worth, and Mrs. Edith Heron and Mrs. Ethel Williams, both of Lovington, N. Mex.

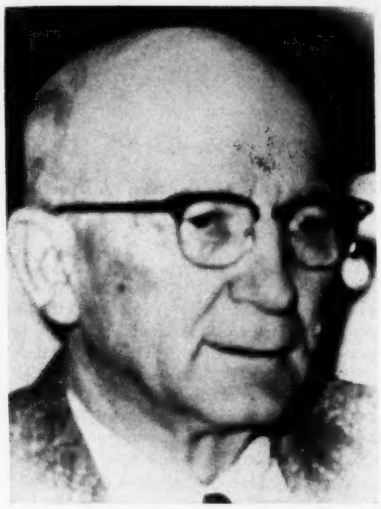
DR. DAN A. RUSSELL

Dr. Daniel Abell Russell, eye, ear, nose and throat specialist in San Antonio since 1922, died February 2, 1957, at a San Antonio hospital.

Dr. Russell was born October 22, 1895, in Bexar County, and his parents were Fred R. and Hulda (Benke) Russell. After attending public schools in San Antonio, he spent three years at the University of Texas. He then entered Tulane University School of Medicine, New Orleans, from which he was graduated in 1921. He was a member of the United States Naval Reserve Force, Hospital Apprentice, stationed in New Orleans while a medical student. After graduation he interned at Santa Rosa Hospital, San Antonio. Dr. Russell was associated with Dr. D. T. Atkinson, San Antonio, for 11 years, then set up his own private practice. Three years ago his son, Dr. Dan A. Russell, Jr., became associated with him. He took postgraduate study at the New York Polyclinic Medical School and Hospital; Washington University Medical School, St. Louis; and George Washington University School of Medicine, Washington, D. C.; and studied under Dr. Chevalier Jackson at Temple University, Philadelphia. He was a fellow of the American Academy of

Ophthalmology and Otolaryngology and a diplomate of the American Board of Otolaryngology.

Dr. Russell was a member of the Texas and American Medical Associations through the Bexar County Medical Society; past president of the San Antonio Ophthalmological and Otolaryngological Society; and past vice-president of the Texas Society of Ophthalmologists and Otolaryngologists. Also, Dr. Russell was associate professor of otolaryngology at the San Antonio branches of the University of Texas and Baylor University medical schools; a civilian consultant in ear, nose, and throat at Brooke Army Hospital; and consultant for Santa Rosa Hospital, San Antonio; Peterson Memorial Hospital, Kerrville; and the New Braunfels Hospital.



DR. DAN A. RUSSELL

Formerly he had served as chief of ear, nose, and throat services at Santa Rosa and Robert B. Green Hospitals for many years. He was vice-president of Phoenix Refining Company, San Antonio, and was a member of the San Antonio Country Club. Hunting, fishing, and poker were hobbies that Dr. Russell enjoyed.

He was married November 21, 1921, in San Antonio to Miss Naomi Gouger. She survives him, as do a son, Dr. Dan A. Russell, Jr., San Antonio; two daughters, Mrs. James D. Folbre, San Antonio, and Mrs. Michael Kangerga, Henderson; five grandsons and five granddaughters; and two brothers, Raymond R. Russell and Ralph W. Russell, both of San Antonio.

DR. JAMES H. PARK, JR.

Dr. James H. Park, Jr., died in a Houston, Texas, hospital February 6, 1957.

Dr. Park was born January 30, 1892, in Mexia; his parents were James Howard and Eva (Riggs) Park. He received a degree in pharmacy from Tulane University, New Orleans, and was graduated from Tulane University School of Medicine in 1916. After interning in Touro Infirmary, New Orleans, he spent a year in residency in Massachusetts General Hospital, Boston, where he specialized in pediatrics. He took postgraduate training at Rockefeller Institute Hospital, New York, in 1918, and began practicing his specialty

DR. JAMES H. PARK, JR.

in Houston the following year. When Baylor University College of Medicine was moved to Houston in 1943, Dr. Park was named professor of pediatrics and chairman of the department, which he organized. In 1947, he became clinical professor of pediatrics. In recognition of his fine work, the Pathology Conference Room at Baylor was furnished in Dr. Park's honor by his friends in 1950.

He was a member of the Texas and American Medical Associations through the Harris County Medical Society, and had served as chairman of the Section on Pediatrics in 1947. He was a fellow of the American College of Physicians and a diplomate of the American Board of Pediatrics, and held membership in the Association of American Physicians and Surgeons, American Academy of Pediatrics, Texas Pediatrics Society (of which he was a past president), Houston Pediatrics Society, Alpha Omega Alpha honorary medical fraternity, Methodist Church, Masonic Lodge, and Shrine. He was a former president of the Memorial Hospital staff in Houston, and enjoyed hunting, fishing, and golf as his hobbies.

During World War I, he held the rank of first lieutenant in the Army Medical Corps.

Miss Edith Brownell and Dr. Park were married in Columbia, S. C., on May 14, 1919. Mrs. Park survives as do two daughters, Mrs. W. M. Wheless, Jr., and Mrs. J. V. Neuhaus III; two sons, Dr. David R. Park and James H. Park III, and 11 grandchildren, all of Houston.

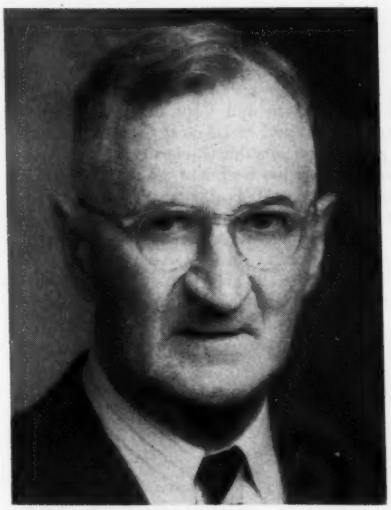
DR. WILLIAM R. JAMIESON

Dr. William Ross Jamieson, El Paso, Texas, died of arteriolar nephrosclerosis January 24, 1957, in a hospital in Fort Bayard, N. Mex.

Dr. Jamieson, son of William A. and Isabel (Ross) Jamieson, was born in Ottawa, Ontario, Canada, February 6, 1875. He attended schools in Ottawa, and was graduated from high school at the age of 14. Because of his youth, he was refused admission to McGill University Medical School, Montreal, so he worked four years in his father's drugstore. He then entered medical school, and was graduated in 1898. He set up his practice in Oklahoma, then

Indian Territory. In 1901, he moved to Torreon, Coahuila, Mexico, to practice 10 years before the outbreak of the Mexican Revolution. At that time, he moved to El Paso, where he practiced until the time of his death.

He had served as chief of staff of El Paso General Hospital, and was a member and past president of the El Paso County Medical Society, Reserve Officers Association, and Southwestern Medical Association. He was the only man who had twice been president of the latter group. He also was a member of the staff of Hotel Dieu, the American Urological Association, American Medical Association, and



DR. WILLIAM R. JAMIESON

Texas Medical Association, in which he held honorary membership. He was a member of the Masonic Lodge and Rotary International.

During World War I, Dr. Jamieson was a major in the Army Medical Corps and organized a field hospital unit. He was a retired colonel in the Army Medical Corps Reserve.

Miss Tulitas Wulff and Dr. Jamieson were married in San Antonio April 5, 1906. She survives, as do a sister, Mrs. Eva Gray, and two brothers, Leslie Jamieson and Jack Jamieson, all of Ottawa.

DR. HENRY PHILIPS

Dr. Henry Fuller Philips, Col., USA (Ret.), Dallas, Texas, died March 4, 1957, in the Veterans Administration Hospital at Lisbon.

Born near Nashville, Tenn., May 11, 1881, Dr. Philips attended the public schools in Tyler, Texas. He was graduated from the University of Texas School of Medicine in 1907 and served an internship at John Sealy Hospital, Galveston. After practicing briefly in Big Spring, then his home town, he joined the United States Army in 1908, being sent first to the Army Medical School in Washington, D. C. He then was at Fort Clark, Texas, for a short while before being transferred to the Philippine Islands for two years. After an interim period on the Texas-Mexican border with headquarters at El Paso, he returned to the Philippines for four years. In 1918 he was sent with the Army to Siberia for a year. Next he was stationed at Laredo, then at Fort Sam Houston, next at Fitzsimons Army Hospital, Denver, and then at Beaumont Army Hospital, El Paso. After five years as medical ROTC instructor at Baylor University College of Medicine, at that time in Dallas, Dr. Philips served at Edgewood Arsenal, Md., and Fort Ontario, N. Y. He then requested retirement because of length of service. The request was granted, but owing to upset world conditions then prevailing, Dr. Philips offered to continue on active duty and was sent again to Baylor with the ROTC. He suffered a heart attack in class in 1943, and after a period of hospitalization was retired for physical disability November 30, 1944.

After his retirement, Dr. Philips made his home in Dallas, where he was an active member of the board of deacons of Gaston Avenue Baptist Church and was a Mason. Dr. Philips was a service member of the American Medical Association.

Surviving Dr. Philips is his wife, the former Miss Lois Allen of Lampasas, whom he married September 23, 1908. Other survivors are two daughters, Mrs. A. E. C. Pope of Cedar Valley and Mrs. P. L. Barnes of Winston-Salem, N. C.; two brothers, Shine Philips of Big Spring and D. D. Philips, D.D.S., of Mount Pleasant, Tenn.; one sister, Mrs. B. D. Boswell of El Paso; five grandchildren; and two great grandchildren.

DR. STEVE F. TURNER

Dr. Steve Francis Turner died in his home in El Paso, Texas, on February 2, 1957.

He was born in Commerce, Texas, on December 3, 1911, the son of Tom L. and Lida Jack Turner. A graduate of Paris, Texas, High School, he was graduated from Paris Junior College in 1930, attended East Texas State Teachers College in Commerce, and Southern Methodist University in Dallas. He received his medical degree in 1935 from the University of Texas Medical Branch in Galveston.

Dr. Turner interned at St. Elizabeth's Hospital in Dayton, Ohio. He practiced briefly in Paris; with CCC camps in Dyess Colony, Oark and Russellville, Ark.; and in El Paso from 1941 to 1957. He was owner of the Turner Clinic in El Paso.

DR. STEVE F. TURNER

He was a member of Lamar and El Paso County Medical Societies, Texas Medical Association, and the American Medical Association. In addition, he was a judge and member of the American Kennel Club; member of the American Orchid Society, Knife and Fork Club, El Paso Junior Chamber of Commerce, and Montana Street Church of Christ. He was a philatelist and artist.

Dr. Turner is survived by his mother, Mrs. Tom Turner of Paris; a sister, Mrs. Lida Rolan Lewis of Odessa; and one nephew and two nieces who were his foster children, Tom Tandy Lewis, Madge Frances Lewis, and Millie Sue Lewis, all of Odessa.

DR. HUGH J. STENNIS

Dr. Hugh Joseph Stennis, Abilene, Texas, died January 16, 1957, of injuries received in an automobile accident north of Brownwood.

Dr. Stennis was born in Carlsbad, N. Mex., June 27, 1922; his parents were Samuel D. and Jeannie (McLees) Stennis. After completing his preliminary education at Pampa High School, Dr. Stennis attended the Schreiner Institute, Kerrville, and the University of Texas, Austin, for one year each, then interrupted his schooling to serve in the United States Navy during World War II. He was graduated in 1945 from Southern Methodist University, Dallas, and received his medical training at the University of Pennsylvania Medical School, Philadelphia. In 1949-1950, he interned at Hermann Hospital, then served two years as a captain in the Army Air Force Medical Corps and was stationed at Ellington Field, Houston. While in the service, he took postgraduate work in preventive medicine at Tulane University School of Medicine, New Orleans.

Dr. Stennis began his medical practice in Sweetwater in 1953, and was county health officer of Nolan and Scurry Counties for a year. He then moved to Abilene, where he

DR. HUGH J. STENNIS

was county health officer at the time of his death. In 1956, he earned his master of arts degree in public health from the Harvard Graduate School of Medicine, Boston.

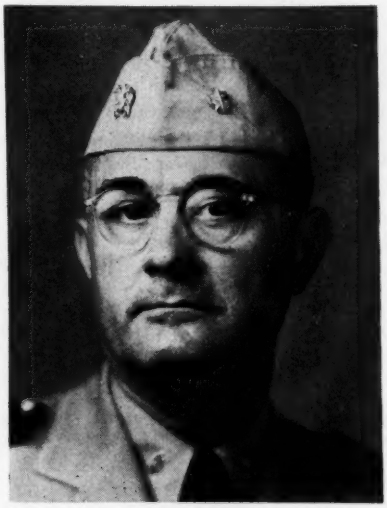
Dr. Stennis had been a member of the Texas and American Medical Associations through the Taylor-Jones Counties Medical Society, and was vice-president of his local mental health society.

Miss Shirley Beatrice Berryman and Dr. Stennis were married March 10, 1950, in Houston. Survivors include Mrs. Stennis and two sons, Richard F., age 5, and Charles L., age 3, all of Houston; his mother, Mrs. S. D. Stennis, Pampa; two brothers, S. Y. Stennis, Amarillo, and H. J. Stennis, Dallas; and a sister, Mrs. H. E. Howard, Coleman.

DR. FRED M. HUGHES

Dr. Fred Morris Hughes, Houston, Texas, died of a heart attack January 28, 1957, just after completing a minor operation on one of his patients in a local hospital.

He was born February 26, 1898, in Ozark, Ala., and was the son of Elvin and Cora Hughes. After attending Texas Agricultural and Mechanical College, College Station, he was graduated from the University of Texas, Austin, in 1923, and received his doctor of medicine degree from the University of Texas Medical Branch, Galveston, in 1926. After interning in Hermann Hospital, Houston, he was a resident physician and surgeon there for two years. He began his private practice in Houston, and was active there at the time of his death. He formerly was on the surgical staff of Jefferson Davis Hospital for about 15 years.



DR. FRED M. HUGHES

He was a member of the Harris County Medical Society, Texas Medical Association, American Medical Association, Post Graduate Medical Assembly of South Texas, Theta Kappa Psi medical fraternity, West University Methodist Church of which he was a steward, Masonic Gray Lodge number 329 of which he was a past master, York Rite Masonic organizations, the Shrine, the Order of the Eastern Star, and American Legion for which he was medical officer.

He was in the United States Navy during World War I, and reentered the service in 1942. During World War II, he served at the Corpus Christi Naval Air Base and as a medical officer in the Southwest Pacific. He held the rank of commander when he received a terminal leave in 1945.

February 11, 1928, Dr. Hughes and Miss Ava Mae Bevil of Kountze were married in Houston. She survives, as does one brother, Charles L. Hughes, Houston.